30. 4. 91

UCM

THE BRITISH JOURNAL OF PSYCHIATRY

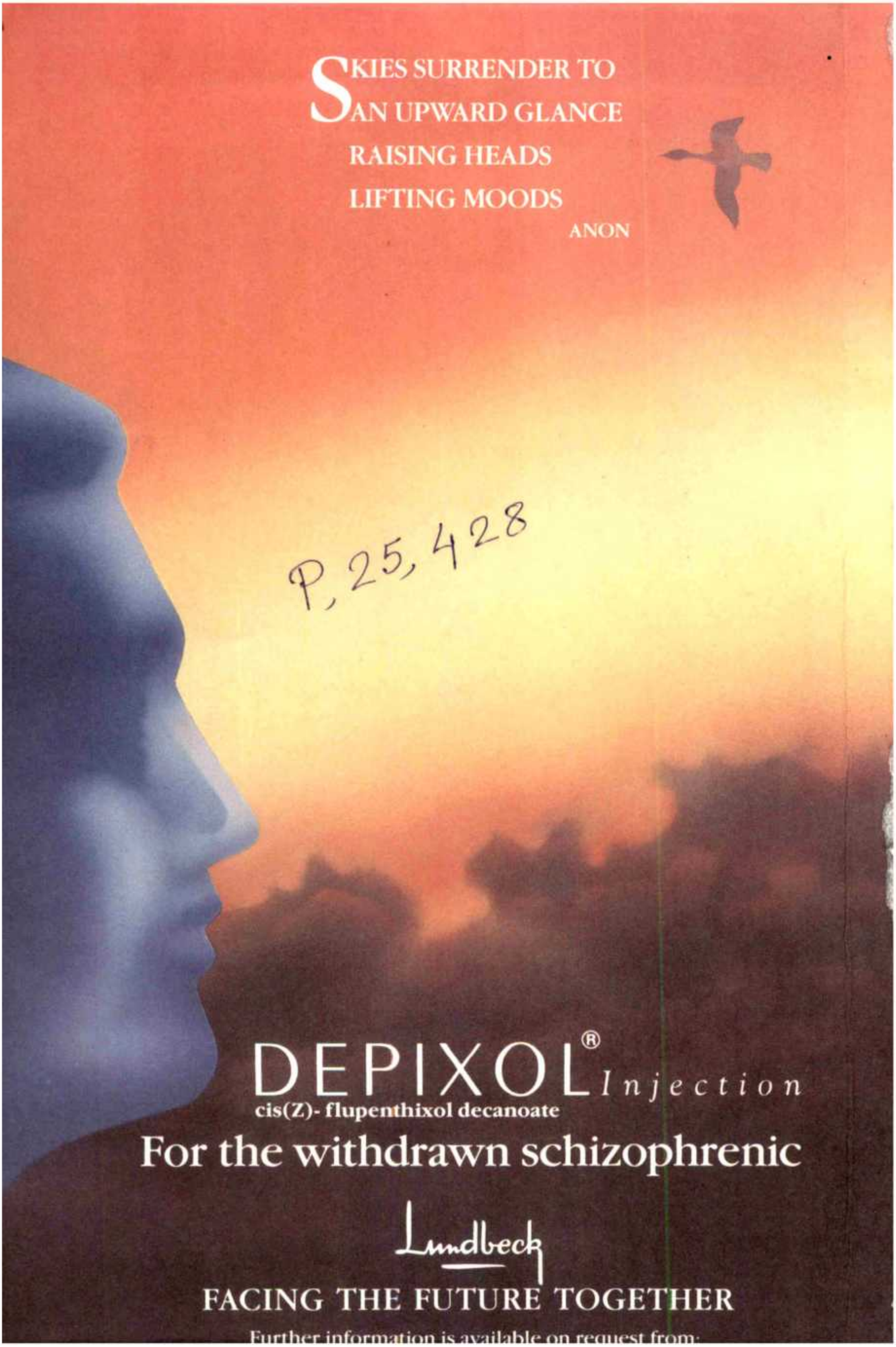
August 1990 Vol 157

40

27

Published by
The Royal College of Psychiatrists

ISSN 0007-1250



Further information is available on request from:

P, 25,428

THE BRITISH JOURNAL OF PSYCHIATRY

August 1990 Volume 157

C0H - HO 6172-27-P25428

Annotation

The (frequently) neuroleptic (potentially) malignant syndrome. *A. M. P. Kellam* 169

Lecture

Educating the psychiatrist of the 21st century. *R. H. Cawley* 174

Papers

Further investigation of the predictors of outcome following first schizophrenic episodes. *E. C. Johnstone, J. F. MacMillan, C. D. Frith, D. K. Benn and T. J. Crow* 182

Alcoholism: a follow-up study of participants in an alcohol treatment programme. *G. K. Shaw, S. Waller, S. McDougall, J. MacGarvie and G. Dunn* 190

The case-control study in psychiatry. *G. Lewis and A. J. Pelosi* 197

^{3}H-imipramine binding to previously frozen platelet membranes from depressed patients, before and after treatment. *D. Healy, A. E. Theodorou, A. M. Whitehouse, K. M. Lawrence, W. White, H. Wilton-Cox, S. M. Kerry, R. W. Horton and E. S. Paykel* 208

Multimodal imaging in Alzheimer's disease. The relationship between MRI, SPECT, cognitive and pathological changes. *J. A. O. Besson, J. R. Crawford, D. M. Parker, K. P. Ebmeier, P. V. Best, H. G. Gemmell, P. F. Sharp and F. W. Smith* 216

Daughters of people with dementia. Expressed emotion, strain and coping. *K. D. Bledin, B. MacCarthy, L. Kuipers and R. T. Woods* 221

Further change in the pattern of mental illness in the elderly. *A. B. Christie and E. R. M. Wood* 228

A double-blind placebo-controlled study of buspirone in diazepam withdrawal in chronic benzodiazepine users. *C. H. Ashton, M. D. Rawlins and S. P. Tyrer* 232

Accounting for delusional misidentifications. *H. D. Ellis and A. W. Young* 239

Factors related to self-reporting of the pre-menstrual syndrome. *P. Warner and J. Bancroft* 249

A controlled study of psychological and social change after surgical gender reassignment in selected male transsexuals *C. Mate-Kole, M. Freschi and A. Robin* 261

Clinical features of patients attending a gender-identity clinic. *A. Burns, M. Farrell and J. Christie Brown* 265

The Current Literature

Collected and neglected: are Oxford hostels for the homeless filling up with disabled psychiatric patients? *P. Garety and R. M. Toms* 269

Brief Reports

Is parental age related to the risk of Alzheimer's disease? *A. Hofman, C. M. van Duijn, W. Schulte, T. A. Tanja, R. Haaxma, A. J. Lameris and R. J. Saan* 273

The effects of chronic lithium treatment on psychomotor performance related to driving. *S. Hatcher, R. Sims and D. Thompson* 275

Do-Do abuse. *S. Loosmore and D. Armstrong* 278

A psychiatric study of deviant eating behaviour among mentally handicapped adults. *G. O'Brien and A. M. Whitehouse* 281

Firesetting in an adolescent boy with Asperger's syndrome. *I. P. Everall and A. LeCouteur* 284

The validation of the Edinburgh Post-natal Depression Scale on a community sample. *L. Murray and A. D. Carothers* 288

Folie à trois among two Soviet–Jewish immigrant families to Israel. *S. Maizel, H. Y. Knobler and R. Herbstein* 290

Rabbit syndrome – a rare complication of long-term neuroleptic medication. *D. K. Deshmukh, V. S. Joshi and M. R. Agarwal* 293

Correspondence 294

Books Reconsidered

The Theory and Practice of Group Psychotherapy (Irvin D. Yalom). *E. C. Crouch* 304

Book Reviews 307

American Journal of Psychiatry, Volume 147, Number 7, July 1990 318

The Journal of Nervous and Mental Disease, Volume 178, Number 7, July 1990 *facing* 318

(N.B. With effect from January 1991, the contents list will be moved to the cover of the *Journal*).

DALHOUSIE UNIVERSITY DEPARTMENT OF PSYCHIATRY
DIVISION OF CHILD PSYCHIATRY

Child Psychiatry Vacancy

The Division of Child Psychiatry is seeking a fully qualified child psychiatrist for a full-time academic position. This position includes active staff membership at the Izaak Walton Killam Children's Hospital, Halifax, Nova Scotia.

Academic duties would include teaching undergraduate and postgraduate trainees and participating in continuing medical education. Active research interests would be an asset.

Clinical duties would largely be with outpatients and may include community consultation. Depending on seniority, the incumbent might assume direction of psychiatric outpatient services at the Izaak Walton Killam Children's Hospital. A small private practice is negotiable.

There is an opportunity for further development of an academic child psychiatry service. The child mental health service delivery system in Nova Scotia is under active review and new directions for child psychiatry are being planned with the potential of modernising and expanding these services.

Qualifications for the position are the F.R.C.P.C. or equivalent in psychiatry: full member status in the Canadian Academy of Child Psychiatry; and a current license to practice in Nova Scotia.

"Dalhousie University has a policy of affirmative action in hiring qualified women academic staff and in accordance with Canadian immigration requirements, priority will be given to Canadian citizens and permanent residents."

Interested candidates should write, including an updated curriculum vitae, to: **Dr A. Munro, Head, Dalhousie University Department of Psychiatry, Camp Hill Medical Centre, 4th Floor – Lane Building, 1763 Robie Street, Halifax, Nova Scotia B3H 3G2, Canada.**

Future Workshops at The

NHS TRAINING AND STUDIES CENTRE
THE WHITE HART, HARROGATE

The Future Management Role of the Consultant Psychiatrist
19–21 September 1990

Consultant Psychiatrists Accompanied by Their Manager
31 October–2 November 1990

The Future Management Role of the Consultant Psychiatrist
20–22 February 1991

Management Skills for Senior Registrars in Psychiatry
24–26 April 1991

PROGRAMME LEADERS: **Helena Waters** **John Sturt**

For further information please contact: Mrs Olive Moore, The White Hart, Cold Bath Road, Harrogate HG2 0NF (telephone: 0423 505681).

The British Journal of Psychiatry

August 1990, Volume 157

Published monthly. Founded in 1853 as the *Asylum Journal* and known as the *Journal of Mental Science* from 1855 to 1963

Editor: Hugh Freeman

Senior Associate Editor
Alan Kerr

Associate Editors
Dora Black, Sidney Crown, Julian Leff, Sir Martin Roth, Peter Tyrer

Assistant Editors
Mohammed Abou-Saleh, Peter Bruggen, Patricia Casey, Alexander Kellam, Stuart Montgomery, Catherine Oppenheimer, Ian Pullen, Rosalind Ramsay, Henry Rollin, Oliver Russell, George Stein, Digby Tantam, Greg Wilkinson, Stephen Wilson

Publications Manager
Dave Jago

Scientific Editor
Ralph Footring

Bulletin Manager
Elaine Millen

Assistant Scientific Editor
Katy Wimhurst

Editorial Assistants
Judy Ashworth, Ian O'Reilly

⊛™ The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences—Permanence of Paper for Printed Library Materials. ANSI Z39. 48–1984.

Printed by Henry Ling Ltd, The Dorset Press,
23 High East Street, Dorchester, Dorset DT1 1HD
(tel. 0305-251 066).

©1990 The Royal College of Psychiatrists

Subscriptions
Non-members of the College should contact the British Journal of Psychiatry Subscription Department, The Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. Annual subscription rates for 1990 (12 issues post free) are as follows:

UK: institutions £105, individuals £86
US: institutions $265, individuals $162
Elsewhere: institutions £132, individuals £106
Single copies of the Journal, £10, $20 (post free)

Payment should be made out to the British Journal of Psychiatry.

Queries from non-members about missing or faulty copies should be addressed within six months to the same address; similar queries from College members should be addressed to the Registration Department, The Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG.

Advertising
Correspondence and copy should be addressed to Peter T. Mell, Advertisement Manager, PTM Publishers Ltd, 282 High Street, Sutton, Surrey SM1 1PQ (tel. 081-642 0162, fax 081-643 2275).

US Mailing Agents: Mercury Airfreight International Ltd. Inc., 2323 Randolph Avenue, Avenel, NJ 07001.

Back issues published before 1988 may be purchased from William Dawson & Sons Ltd, Cannon House, Folkestone, Kent, England (tel. 0303-850-101).

A CANADIAN OPPORTUNITY

MEDICINE HAT REGIONAL HOSPITAL

requires a

PSYCHIATRIST

The Position: To work on a sessional basis as a Psychiatrist on the Adult Psychiatric Services of the Hospital. Will be expected to work closely with the Chief of Psychiatry in developing services in the community.

The Hospital: Medicine Hat Regional Hospital is a district general hospital with a modern 26 bed Psychiatric Unit with a Day Hospital Program. Psychogeriatric and Child Psychiatric Services and provided by other psychiatrists working on sessional basis.

The Location: Medicine Hat is located in the South Eastern corner of Alberta. The Hospital has a catchment area of 79,000. A wide range of recreational facilities are available.

Qualifications: Minimum MRCPsych. Graduates of Medical Schools other than United Kingdom, Australia and New Zealand may require special qualifications.

Visa: Assistance will be provided to the successful candidate towards obtaining the necessary Visa.

Remuneration: Approx. CA$150,000.

Interviews: Preliminary interviews will be held in London, England in September or October.

Apply with CV and names of three references in confidence to:
Dr S. Nandy, Chief of Psychiatry, Medicine Hat Regional Hospital,
666 – Fifth Street S.W., Medicine Hat, Alberta, Canada T1A 4H6
Tel. 403-529-8036: Fax. 403-529-8010.

ST GEORGE'S HOSPITAL
MEDICAL SCHOOL
(University of London)

Part-time MSc in Clinical Psychotherapy

The Psychotherapy Section of the Academic Department of Mental Health Sciences, St George's Hospital Medical School, is offering the MSc course in Clinical Psychotherapy (University of London) to commence in October 1990. The course will last for three years on a one-day per week basis, and will cover theoretical and practical components of individual, group, family and behavioural psychotherapies. Course fees will be £993 per annum but will be subject to reduction in the case of employees of the Wandsworth Health Authority and certain other applicants. All interested medical practitioners should please apply for further information as soon as possible from Mrs Jane Frisk, Psychotherapy Section, Academic Department of Mental Health Sciences, St George's Hospital Medical School, Cranmer Terrance, Tooting, London SW17 0RE. Telephone: 081 672 9944, ext 56020.

NOTICE TO ADVERTISERS

Applications for advertisement space in *The British Journal of Psychiatry* should be made to:

PTM PUBLISHERS LIMITED

282 High Street,
Sutton, Surrey SM1 1PQ

Telephone: 081-642 0162
Fax: 081-643 2275

British Journal of Psychiatry (1990), **157**, 169–173

Annotation

The (Frequently) Neuroleptic (Potentially) Malignant Syndrome

A. M. P. KELLAM

Since a previous review (Kellam, 1987), the subject of neuroleptic malignant syndrome (NMS) has continued to cause considerable interest, particularly in the USA where there is still controversy about its frequency, despite two attempts to survey prospectively all cases admitted to two different hospitals. Cases of NMS are now being more widely recognised by non-psychiatrists, especially geriatricians.

Evidence continues to amass from case reports that the administration of oral bromocriptine or other dopamine agonists, with intravenous dantrolene, if necessary, is associated with a survival rate much higher than in cases treated by supportive measures only.

There is also continuing interest in the boundaries of NMS and its relationship both to catatonia, including the acute lethal subtype, and to borderline cases where there is confusion and either fever or Parkinsonian rigidity. The concept that NMS is the extreme end of a spectrum starting from normal neuroleptic-induced Parkinsonism has been put forward but Adityanjee *et al* (1988) argue against this approach and stress the advantages of strict diagnostic criteria.

Review of the literature

Incidence of NMS

Addonozio *et al* (1986), in a retrospective survey of 82 consecutive in-patients, found the highest incidence of NMS yet claimed – 2.4% of cases treated with neuroleptics – and suggested that if partial cases were included the rate could be as high as 12.2%. A retrospective survey of patients admitted and treated with neuroleptics over one year by Pope *et al* (1986) found an incidence of 1.4%: eight out of 483 patients experienced 11 episodes of definite (six) or probable (five) NMS. My own criteria (Kellam, 1987) would have excluded two of the 11, possibly three, due to the absence of pyrexia over 37.5°C. A subsequent prospective work by the same authors (Keck *et al*, 1987), surveying 679 consecutive admissions treated with neuroleptics, found six cases, an incidence of around 1%. They excluded two cases, one as apyrexial, and the other as not having had neuroleptics for six months because of previous NMS. This patient was on lithium only. Two other patients who were known to have had NMS previously were admitted and treated without using neuroleptics.

A far lower incidence of 0.07% was found by Gelenberg *et al* (1988), who prospectively surveyed 2173 admissions and identified only one patient who developed NMS out of 1470 who were given neuroleptics. Even this rate appears far higher than that seen in the UK, but there is an urgent need to carry out a prospective survey on a large series of psychiatric patients admitted to hospital in Britain and treated with neuroleptics, to try and establish whether the rate is indeed so low, or whether, as some people fear, a large percentage of the cases are being missed. All that would be required is for every patient admitted, who was treated with neuroleptics, to have his/her temperature recorded, along with the presence of any rigidity or other Parkinsonian symptoms, at least daily (preferably twice daily). Doubtful cases could then have serum creatinine phosphokinase (CPK) estimations, and could also be examined for level of consciousness. Such a study would also shed light on the occurrence of partial syndromes, revealing whether they are as common as suggested by Addonozio *et al* (1986).

No one has yet related any of these incidence rates to a general population, or offered any estimate of the incidence of neuroleptic prescriptions among them.

Aetiological factors

NMS has now been reported with all the neuroleptics in use, including the substituted benzamides (e.g. sulpiride), loxapine and zuclopenthixol decanoate. Metoclopramide has been implicated twice; this is a drug closely resembling chlorpromazine in its actions, although it is normally used as an antiemetic and not as a neuroleptic. NMS is also reported regularly with the use of other drugs which alter dopaminergic function, both with the cessation of dopamine agonists and the administration of antidepressants (tricyclic as well as monoamine oxidase inhibitors). NMS has also been associated with amoxapine (a newer tricyclic antidepressant) in the USA. Recently, a somewhat similar syndrome, only without Parkinsonism, has been noted in cocaine abuse (Kosten & Kleber, 1988).

The drugs reported to have caused NMS, which might least be expected to affect the brain dopaminergic systems, are clozapine (a new and atypical antipsychotic, which is virtually free from extrapyramidal side-effects and activity involving central dopaminergic systems), carbamazepine, and lithium alone or with clozapine. Possible mechanisms for lithium-induced NMS are discussed by Susman & Addonozio (1987).

Other possible aetiological factors considered recently are external heat stress – Shalev *et al* (1988) reported three cases occurring during a heat wave – and a family history of malignant hyperthermia, although the latter was found not to be an important factor by Hermesh *et al* (1988) who reported no cases of it among either 20 patients who had had NMS or their 108 relatives. The patients themselves had between them undergone 12 operations and 20 courses of electroconvulsive therapy (ECT), while 37 of their relatives had also undergone operations or ECT without any malignant hyperthermia.

The occurrence of NMS in geriatric patients was reported by Finucane *et al* (1988), and a case associated with Alzheimer's disease described by Serby (1986). NMS in the elderly has been reviewed by Addonozio (1987); presumably, this simply reflects the frequent use of neuroleptics to control confusion and agitation in elderly patients but the possibility that brain damage predisposes to the development of NMS must also be considered.

Diagnostic and conceptual issues

A series of cases reported by Barnes *et al* (1986), under the title of 'the syndrome of Karl Ludwig Kahlbaum', discussed catatonia from a neurological standpoint. Some of the cases appeared to fill most of the criteria for NMS and this is especially interesting, as some of these patients, possibly even some of those who clearly suffered from schizophrenia, did not appear to have been treated with neuroleptic agents. However, whether any of them had fever, and therefore met the full criteria for NMS, is not made plain.

The relationship of NMS to lethal catatonia is discussed by Mann *et al* (1986) who conclude that lethal catatonia still occurs and that NMS may be considered as a neuroleptic-induced iatrogenic form. By contrast, Levinson & Simpson (1986), considering 39 cases of NMS, felt they could identify three groups within it: those with concurrent medical problems that could cause the fever, those with medical problems less clearly related to the fever, and those without other medical disorders (14 cases). They concluded that the cases of extrapyramidal symptoms with fever were too heterogeneous to justify the assumption of a unitary 'malignant' syndrome.

Recently, Castillo *et al* (1989) stressed that lethal catatonia begins with psychotic excitement leading to exhaustion, whereas NMS begins with muscular rigidity. Their illustrative case of lethal catatonia however dates from the 1930s. Also, the comparison case of NMS from the 1980s started with confusion and agitation, rigidity only being mentioned later after the fever had started. Coquet-Le Pape (1989) reported a case clearly resembling NMS in a patient who had stopped neuroleptics three months previously. She compared it with a definite case of NMS and no distinguishing features emerged. That NMS can be produced in non-psychotic patients by reducing their dopaminergic drive is clear (Kellam, 1987). That a syndrome identical to NMS can occur in schizophrenic patients in the absence of neuroleptics seems also certain and paradoxical, as here the dopaminergic drive is believed to be overactive.

The opposite point of view was adopted by Rosebush & Stewart (1989) who prospectively studied 24 cases in 20 patients between 1981 and 1987 and found that only one was diagnosed as suffering from schizophrenia, 14 had affective disorders and 42% of the cases had other brain pathology. They felt catatonia could easily be distinguished and that catatonic patients would have worsened rather than improved when neuroleptics were stopped. They also commented that serum iron was reduced below the normal level in 18 of 19 patients in whom it was measured, a finding never recorded before. All their cases survived and there were few sequelae, most with only supportive care.

The spectrum concept of NMS, that it shades imperceptibly into the ordinary extrapyramidal reactions to neuroleptics, appears to have first been made by Fogel & Goldberg (1985). Possibly, as Adityanjee *et al* (1988) suggest, this has been responsible for the high incidences reported from the USA because borderline cases have been included. They suggest very strict criteria, including a fever in excess of 39.0°C, clouding of consciousness, autonomic dysfunction, and rigidity, as all being necessary for the diagnosis, while accepting that partial and aborted forms also exist. Certainly, these strict criteria would define severe forms where the patient's life is in danger unless urgent treatment is instituted, but they might equally exclude cases demonstrating how the syndrome develops and how attacks can be aborted.

Treatment

Treatment appears to have made a fatal outcome now unusual. A review by Shalev *et al* (1989), of 202 cases published between 1959 and 1987, showed a mortality of 18.8%, which reduced to 11.6% when only the cases after 1984 were considered. Interestingly haloperidol was associated with a lower mortality. Finally, they recommended that the life-threatening complications, rhabdomyolisis, myoglobinaemia, and renal failure, can most easily be watched for by daily CPK estimations, which reflect the degree of muscle breakdown.

In addition to the immediate cessation of neuroleptics, most authors recommend the administration of a dopamine agonist, usually oral bromocriptine (5–10 mg, t.d.s.). Rapid improvement, within three hours, is usual. Unfortunately, the drug can only be given orally, and if a nasogastric tube is employed, there is sometimes a danger of aspiration. Dantrolene sodium, a skeletal muscle relaxant, can be given intravenously in doses of 50 mg every 12 hours for up to seven doses. However, prolonged administration carries the risk of hepatotoxicity, and therefore it is probably best used in cases where oral bromocriptine cannot be given because of the rigidity, and should be replaced by it as soon as the patient is sufficiently relaxed to take drugs by mouth. There is a good review of all aspects of the diagnosis and treatment by Tesar (1988) and detailed reports of the use of dantrolene and three dopamine agonists by Lazarus (1986*b*). As well as bromocriptine, the other dopamine agonists used were oral amantadine (100 mg b.d.) (Hamburg *et al*, 1986) and L-dopa in the same dosage (Knezevic *et al*, 1984). Rosenberg & Green (1989) studied 64 treated cases from the world literature and concluded that with supportive measures alone, the mean time to a clinical response was 6.8 days, while the addition of dantrolene reduced this to 1.2 days and bromocriptine to 1.0 days.

A remaining problem is treatment of a continuing or recurring psychosis, after the successful treatment of NMS. Hermesh *et al* (1987) recommended ECT, but Adityanjee (1987) argued that further neuroleptic treatment is safer. ECT appears to be safe, however, *after* the syndrome has subsided (Lazarus, 1986*a*) although not *during* the syndrome itself, when fatalities have been recorded (Adityanjee, 1987). Another approach has been to use bromocriptine and thioridazine simultaneously, controlling problems and monitoring for the recurrence of NMS by means of regular estimations of the CPK level (Goldwasser *et al*, 1989).

The probability of recurrence of NMS when a recovered patient is 'challenged' by further neuroleptic treatment is discussed by Wells *et al* (1988). They review both a case where NMS recurred and the literature of a further 40 patients, three of whom were rechallenged twice (44 challenges in total reviewed). Among the 24 definite cases challenged, six definitely developed NMS again and four probably did. Of 20 probable cases of NMS (mainly fever not reaching 38.0°C) only one definitely developed NMS on challenge, with six possible cases. Whether or not the challenge was with the same neuroleptic, one of similar chemical structure, or a neuroleptic not structurally related to the original did not appear to make any difference. A 'drug holiday' of more than five days before challenge with a further dose of neuroleptic was, however, related to a reduced chance of recurrence. They concluded that further neuroleptic treatment was an acceptable risk, if further treatment was needed.

A similar study by Rosebush *et al* (1989), who studied 20 patients prospectively over six years, reached similar conclusions. Fifteen of the patients were challenged, and in five symptoms recurred; four of these were rechallenged a second time and symptoms recurred in two; finally one of these two was rechallenged a third time without symptoms recurring. Analysing these 20 challenges, a period of over two weeks before challenge or rechallenge was significantly associated with success ($P<0.001$), whereas the associations between success and lower potency or dosage at the challenge did not reach statistical significance. Single case reports include the successful use of molindone after trifluoperazine and thioridazine had caused NMS (Slack & Stoudemire, 1989), and loxapine after NMS had followed the use of trifluoperazine, methotrimeprazine and lithium, and recurred with lithium and carbamazepine (Padgett & Lipman, 1989).

There are now a few follow-up studies of NMS. Rosebush & Stewart (1989) followed up 24 cases (20 patients) for six years, and all survived. Nine had concurrent physical illnesses and dehydration was present in 22. The value of dantrolene and bromocriptine was not supported but they felt that their patients experienced profound anxiety during the attack and recommended benzodiazepines.

Meta analysis

My personal collection of cases and references to NMS from the literature has now grown to 536 entries in a computer data-base. Of these, 421 are cases gathered from 264 papers and other communications. Not all cases had information on every feature. Table I shows the distribution of some cases by age and sex. This cannot be directly compared with the data in Kellam (1987) as the age-groups produced by the computer differ. However, the excessive representation of young men, especially those between the ages of 20 and 40 years, remains

TABLE I

Distribution of 371 cases of NMS by age and sex

Age: years	*0–9*	*10–19*	*20–29*	*30–39*	*40–49*	*50–59*	*60–69*	*70–79*	*80–89*	*90+*
Sex										
male	4	21	65	51	30	18	17	8	3	–
female	2	18	26	23	26	26	19	5	–	1

obvious. The total number of cases over 60 years-of-age has increased to 61, compared with 12 over the age of 65 previously. Indeed, there are now 19 cases over 70 years-of-age.

Peak temperature remains closely associated with survival; all 38 patients whose maximum temperatures were between 37.5 and 37.9°C survived; between 38.0 and 38.9°C, 6 patients died out of 92; between 39.0 and 39.9°C, 10 patients died out of 85; between 40.0 and 40.9°C, 18 out of 79; between 41.0 and 41.9°C, 17 out of 55; and finally over 42°C – which were the highest temperatures recorded – 14 patients died out of 25.

The relationship of diagnosis of patients with NMS to outcome now appears less clear. Of 49 patients given organic diagnoses, 15 died (31%); of 184 with schizophrenia, 30 died (16%); of 137 with affective psychoses, 31 died (23%); of 31 with unspecified psychoses, 6 died (19%); and of 21 with Parkinson's disease, 5 died (24%). (Some cases had more than one diagnosis.) Of 31 with other diagnoses, 12 died (39%). The last figure may well be due to the disporportionate inclusion of old cases from the days when these conditions were virtually untreatable as well as the fact that only spectacular cases ending in death were likely to have been felt worth recording.

The outcome of treatments also change little when new cases are added. Treatment with dantrolene alone was recorded in 44 cases and failed in seven (16%); with bromocriptine alone, treatment failed in five cases out of 42 (12%); with both drugs, treatment failed in three cases out of 22 (14%).

Amantadine was used in 16 cases, with no failures, and L-dopa in 16, with only one failure; these apparent successes may, however, have been due to the other treatments (especially bromocriptine combined with dantrolene) having been used in the more desperate cases. With support alone, 29 patients died out of 109, giving a baseline mortality of 27%, but again, these figures probably include all the old cases. Other treatments recorded include benzodiazepines in 31 cases, and ECT in 15 cases (one patient died). Anticholinergic anti-Parkinsonian agents were used in 17 cases (four patients died), and treatment was not mentioned in 129 cases. (Again, some cases will have been counted twice.)

References

Adityanjee, (1987) Role of electroconvulsive therapy in neuroleptic malignant syndrome. *Acta Psychiatrica Scandinavica*, **76**, 603–604.

——, Singh, S., Singh, G., *et al* (1988) Spectrum concept of neuroleptic malignant syndrome. *British Journal of Psychiatry*, **153**, 107–111.

Addonozio, G (1987) Neuroleptic malignant syndrome in elderly patients. *Journal of the American Geriatric Society*, **35**, 1011–1012.

——, Susman, V. L. & Roth, S. D. (1986) Symptoms of neuroleptic malignant syndrome in 82 consecutive inpatients. *American Journal of Psychiatry*, **143**, 1587–1590.

Barnes, M. P., Saunders, M., Walls, T. J., *et al* (1986) The syndrome of Karl Ludwig Kahlbaum. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 991–996.

Castillo, E., Rubin, R. T & Holsboer-Trachsler, E. (1989) Clinical differentiation between lethal catatonia and neuroleptic malignant syndrome. *American Journal of Psychiatry*, **146**, 324–328.

Coquet-Le Pape, B. (1989) Catatonie aigue et syndrome malin des neuroleptiques. These pour le Diplome d'Etat de Docteur en Medicine. Unpublished thesis, University of Rennes.

Finucane, P., Price, C. & Ghose, K. (1988) Neuroleptic malignant syndrome in an elderly patient. *British Medical Journal*, **296**, 18.

Fogel, B. S. & Goldberg, R. J. (1985) Neuroleptic malignant syndrome (letter). *New England Journal of Medicine*, **313**, 1292.

Gelenberg, A. J., Bellinghausen, B., Wojcik, J. D., *et al* (1988) A prospective survey of neuroleptic malignant syndrome in a short-term psychiatric hospital. *American Journal of Psychiatry*, **145**, 517–518.

Goldwasser, H. D, Hooper, J. F. & Spears, N. M. (1989) Concomitant treatment of neuroleptic malignant syndrome and psychosis. *British Journal of Psychiatry*, **154**, 102–104.

Hamburg, P., Weilburg, J. B., Cassem, N. H., *et al* (1986) Relapse of neuroleptic malignant syndrome with early discontinuation of amantadine therapy. *Comprehensive Psychiatry*, **27**, 272–275.

Hermesh, H., Aizenberg, D. & Weizman, A (1987) A successful electroconvulsive treatment of neuroleptic malignant syndrome. *Acta Psychiatrica Scandinavica*, **75**, 237–239.

Hermesh, H., Aizenberg, D., Lapidot, M., *et al* (1988) Risk of malignant hyperthermia among patients with neuroleptic malignant syndrome and their families. *American Journal of Psychiatry*, **145**, 1431–1434.

Keck, P. E., Pope, H. G. & McElroy, S. L. (1987) Frequency and presentation of neuroleptic malignant syndrome: a prospective study. *American Journal of Psychiatry*, **144**, 1344–1346.

Kellam, A. M. P. (1987) The neuroleptic malignant syndrome, so-called. *British Journal of Psychiatry*, **150**, 752–759.

Knezevic, W., Mastaglia, F. L., Lefroy, *et al* (1984) Neuroleptic malignant syndrome. *Medical Journal of Australia*, **140**, 28–30.

Kosten, T. R. & Kleber, H. D. (1988) Rapid death during cocaine abuse; a variant of the neuroleptic malignant syndrome? *American Journal of Drug and Alcohol Abuse*, **14**, 335–346.

Lazarus, A. (1986*a*) Treatment of neuroleptic malignant syndrome with electroconvulsive therapy. *Journal of Nervous and Mental Disorders*, **174**, 47–49.

—— (1986*b*) Therapy of neuroleptic malignant syndrome. *Psychiatric Developments*, **1**, 19–30.

Levinson, D. F. & Simpson, G. M. (1986) Neuroleptic-induced extrapyramidal symptoms with fever. *Archives of General Psychiatry*, **43**, 839–847.

Mann, S. C., Caroff, S. N., Bleier, H. R., *et al* (1986) Lethal catatonia. *American Journal of Psychiatry*, **143**, 1374–1381.

Padgett, R. & Lipman, E. (1989) Use of neuroleptics after an episode of neuroleptic malignant syndrome. *Canadian Journal of Psychiatry*, **34**, 323–325.

Pope, H. G., Keck, P. E. & McElroy, S. L. (1986) Frequency and presentation of neuroleptic malignant syndrome in a large psychiatric hospital. *American Journal of Psychiatry*, 143, 1227–1233.

Rosebush, P. & Stewart, T. (1989) A prospective analysis of 24 episodes of neuroleptic malignant syndrome. *American Journal of Psychiatry*, 146, 717–725.

——, —— & Gelenberg, A. J. (1989) Twenty neuroleptic rechallenges after neuroleptic malignant syndrome in 15 patients. *Journal of Clinical Psychiatry*, 50, 295–298.

Rosenberg, M. R. & Green, M. (1989) Neuroleptic malignant syndrome. *Archives of Internal Medicine*, 149, 1927–1931.

Serby, M. (1986) Neuroleptic malignant syndrome in Alzheimer's disease. *Journal of the American Geriatric Society*, 34, 895–896.

Shalev, A., Hermesh, H. & Munitz, H. (1988) The role of external heat load in triggering the neuroleptic malignant syndrome. *American Journal of Psychiatry*, 145, 110–111.

——, —— & —— (1989) Mortality from neuroleptic malignant syndrome. *Journal of Clinical Psychiatry*, 50, 18–25.

Slack, T & Stoudemire, A. (1989) Reinstitution of neuroleptic treatment with molindone in a patient with a history of neuroleptic malignant syndrome. *General Hospital Psychiatry*, 11, 365–367.

Susman, V. L. & Addonozio, G. (1987) Reinduction of neuroleptic malignant syndrome by lithium. *Journal of Clinical Psychopharmacology*, 7, 339–341.

Tesar, G. E. (1988) Neuroleptic malignant syndrome an update. *Problems in Critical Care*, 2, 149–158.

Wells, A. J., Sommi, R. W. & Crismon, M. L. (1988) Neuroleptic rechallenge after neuroleptic malignant syndrome; case report and literature review. *Drug Intelligence and Clinical Pharmacy*, 22, 475–480.

Alexander M. P. Kellam, MD, FRCPsych, *Consultant Forensic Psychiatrist, Whitchurch Hospital and The University Hospital of Wales, Heath Park, Cardiff CF4 4XW, Wales*

British Journal of Psychiatry (1990), 157, 174–181

Lecture

Educating the Psychiatrist of the 21st Century

R. H. CAWLEY

Psychiatry is under threat from developments within mental health care. In educating future psychiatrists, more emphasis should be placed upon: collaborative research; integrated teamwork, without interprofessional rivalry; work in primary care; the non-scientific components of psychiatry, centred upon relationships with patients; less dogmatic attitudes for or against psychoanalysis; psychiatry as a branch of medicine; and modern management and audit methods. Younger psychiatrists should be encouraged to take responsibility for shaping the future of the profession.

Education of psychiatrists is a topic of huge importance. My purpose is to review some of the issues, already evident, which may loom large for the psychiatrist of tomorrow. Already, we are educating psychiatrists of the 21st century. The neophyte of today who follows the traditional path may expect to be well established as a consultant by the year 2000 and thus one of those carrying direct responsibility for education in the subject well into the 21st century. Recognising that the pace of social, cultural, and scientific advance is fast increasing, it behoves us to look as well as we can at the far horizon. Lewis (1947) urged that we should learn to distrust and redraw some of our time-worn maps.

Social and political developments in Britain and elsewhere have caused us to face the prospect of far-reaching changes in the economics and management of mental illness and its treatment. These are indeed momentous issues, but I believe the shape of things to come will be determined by matters of even greater importance. I shall review some of the questions which, it seems to me, we must have in the front of our minds when we address the educational requirements of future generations of psychiatrists.

Psychiatry is under threat

Many knowledgeable people have in the last 15 years called attention to the fact that psychiatry will have to struggle for its survival (e.g. Fuller, 1974; Bleuler, 1982; Clare, 1982; Reich, 1982; Romano, 1982; Pichot, 1983).

Blakemore (1981) called for "a paradigmatic change of thinking among the psychiatric fraternity . . . a willingness to help and communicate with the basic sciences," as necessary for progress; and moreover, he said, "psychiatry must realise that one possible outcome of the advance of knowledge will be the sacrifice of psychiatry as an independent way of thinking within medicine." Rawnsley (1984) drew attention to a range of social and professional processes which place psychiatry in jeopardy. Society's ambivalence to mentally ill people and to those who minister to their needs is one factor which may be central and ineluctable but it is not the whole story.

The crucial question is this: what do psychiatrists do that others cannot do – individually or collectively? Look first at the scientific growing points of our discipline. It can be argued that the neurochemist and pharmacologist, the neuroscientist, the experimental and clinical psychologist, the epidemiologist, and the sociologist or social anthropologist might between them take over the task of advancing the scientific study of mental illness. Now turn to the practice of psychiatry. As a general proposition, and most especially in the light of accepted plans for mentally ill people to be treated in the community by what we now call mental health professionals, what does the psychiatrist do in his/her practice which cannot be achieved by the general practitioner (GP), the clinical neurologist, the psychiatric nurse, the clinical psychologist, the occupational therapist, and the social worker, acting individually or in concert?

Can it be, then, that if psychiatry dwindled as an independent way of thinking within medicine, the growth of knowledge about mental illness would continue undiminished, or even accelerate, and mentally ill people would be treated just as well, or even better? This is the *reductio ad absurdum*, yet in the minds of some of our critics these questions are never far away.

Now all psychiatrists will recognise, in the light of their experience, that there are perfectly valid, logical and sufficient responses to these challenges. Psychiatry is an academic and professional discipline with its own specific clinical and research methods, and strengths, problems and responsibilities. But

---

The 11th Aubrey Lewis Lecture, delivered at the Institute of Psychiatry on 13 December 1989.

how can we justify this standpoint, and ensure that it will be underpinned by the educational process? If we concede that psychiatry is under threat, what are the prerequisites for survival?

Some problems and possible remedies

I propose to try to analyse this many-sided predicament, and look for some possible remedies. I have a list of seven distinct themes: no doubt there could be others.

Theme 1. Recent scientific advances in psychiatry

The scientific advances in the psychiatry of the last two decades have been impressive, and the pace seems to be increasing. We need only mention subject headings such as psychiatric genetics, developmental, neuropathological and neurochemical studies, neuropharmacology, neuropsychology, and neuropsychiatry to remind ourselves how much can be said to illustrate the breadth and length of recent gains in neurosciences: gains which have substance and hold promise. Although many advances in understanding and possibly treating mental illness have stemmed from the work of basic scientists, specific contributions from the psychiatrist have been central, or at least essential, components of this work. The psychiatrist is no mere gatekeeper who allows the neuroscientist to have access to patients and their diagnostic labels. And aside from neurosciences, clinical, epidemiological and taxonomic studies, and investigations of interpersonal and social factors affecting the genesis and outcome of mental illness have led to equal advances in other domains, and psychiatrists have patently had a central role in these achievements.

Detailed review of this whole story would surely provide a good case for psychiatry's standing as a *clinical science* – a term which was given precise definition by the celebrated physician Sir Thomas Lewis (1934) (who had no connection with Sir Aubrey). Clinical science is a rigorous science which identifies problems at the 'bedside', uses observations on healthy people, sick people and dead people, and observations, analyses and experiments in the laboratory, to produce advances and new hypotheses which will have cogency back at the bedside.

The great majority of advances in the clinical science of psychiatry have derived from team-work involving psychiatrists and neuroscientists, social scientists, statisticians, and perhaps other clinicians; and the psychiatric researcher has made large, specific and unique contributions. Two things follow: firstly, it will be more than ever important for the psychiatrist of the 21st century to aspire to become a clinical scientist and so have a good grasp of the associated disciplines, both biomedical and psychosocial; and secondly, working partnerships between the psychiatrist and basic or applied scientists are set to become increasingly necessary to advance, or even understand, the clinical science of psychiatry. Collaborative research has become an essential part of a psychiatric education. It will not be good enough if this experience is enjoyed by only a few selected psychiatrists, in collaboration with neuroscientists alone. The relevance of other disciplines – psychology, epidemiology, and social sciences – cannot be too much emphasised. If appropriate collaboration fails to become the rule, included with rigour in the training of all postgraduates, psychiatry will indeed be in jeopardy. But with the right long-term planning, it must surely be the case that psychiatry will become increasingly powerful as a clinical science. Its power resides in the range and toughness of its problems and the breadth of its methodological bases.

Theme 2. Mental health professionals and the psychiatric team

Now what about clinical practice? The psychiatrist's *raison d'être* is the investigation and treatment of mentally ill people. For this, the psychiatrist alone is not enough. People with other kinds of professional training also have a central role. Today we hardly need to rehearse the distinct and complementary activities of the psychiatric nurse, the clinical psychologist, the social worker, and the occupational therapist. Volunteers or voluntary services may be very much in the picture. Properly arranged teamwork is a powerful way of helping the mentally disordered person, it is a most profitable *modus operandi* for the workers, and it is absolutely necessary for the satisfactory education of psychiatrists. The central principle, in brief, is something like this. Each member of the team has both a unique and a shared role in investigating the patient and the illness or predicament, in making decisions concerning management and in the management process itself. The unique role derives from specific professional training, the shared role from what is seen as ethical and humane and reasonable by a person who has experience of working with mentally ill people. There have been few detailed explorations of the social and professional dynamics of the psychiatric team or, to use the modern idiom, the psychiatric team as a system. Heretofore, matters have worked out more or less satisfactorily according to the availability and inclinations of the team members.

In recent years new and divisive forces have assailed the concerted efforts of the psychiatric team. The professional standing and status of both nurses and social workers have become transformed. They have moved towards independence and are not prepared to base their work on doctors' orders. Likewise, clinical psychology has become well established as a specialist field. Clinical psychologists do not like being at the beck and call of psychiatrists, nor do they wish to restrict their clinical work to the mentally ill, recognising that the wide field of behavioural medicine contains a wealth of incentives and rewards. As a result, there has been a strong tendency towards fragmentation of the efforts of the psychiatric team, with different professions tending to go it alone, working sometimes at cross-purposes.

Of course there are still coherent and strong psychiatric teams in which the members see advantage for the patient and satisfaction for themselves in working closely together. But it is becoming more difficult to establish and maintain such integrated team-work. There has been a call to assert that one or other discipline must 'lead' the team. Who should lead and who should follow? Nobody can win this sort of contest. It is regrettable and, with care and forethought, avoidable. Matters of responsibility and accountability are complicated, but can surely be resolved if the members of the team recognise its collective purpose and strength, and remain aware of how feeble their efforts become if they are not integrated. No effective psychiatric team wastes its time prating about leadership. There is too much else to be done.

If multiprofessional team-work is to survive as a reality and not merely a slogan, initiatives will be needed. These will call for hard work and a very high level of diplomacy. Is it too fanciful to propose that a sensible manifesto for multiprofessional team-work should be developed without delay? Such a declaration would be effective if, and only if, it was the product of joint effort by representative members of *all* the professions involved. It would genuinely help sick people if, and only if, it subsumed a balanced view of what can be done by the best efforts of a multidisciplinary team. If it were predominantly legalistic, professionally defensive, and dominated by general management principles, it would be worse than useless. Some action towards ensuring the survival of effective interprofessional team-work is urgent, and the psychiatrist of the 21st century will be at the centre of the action or the consequences of *laissez-faire*.

Now the incentives for truly collaborative team-work must surely be laid down in the educational process. Trainees, of all the relevant disciplines, are mature people. They are in the best position to see at first hand how the interests of the patient or client, and their own satisfaction in their work, all lie in the stimulus, and indeed privilege, of working with members of their own and other professions. Senior people, of any profession, all too often have practices and outlooks which have become unhappily constrained, stagnant, and unresponsive to change. It is a sad fact that increasing age and length of experience can remarkably weaken the imagination.

Theme 3. Mental disorder is largely a matter for primary care

Nowadays, thanks to the work of Shepherd and his colleagues (Wilkinson, 1989*b*), it is abundantly clear that 95% of patients with mental and emotional disorders are handled by the GP, aided perhaps by other primary health-care workers. They are not seen by psychiatrists. Therefore the psychiatrist, and other members of the team, may have little direct experience of the vast majority of the clinical problems in the field in which they claim expertise. It would be too economical to say that the psychiatrist and those working with him provide the psychosis service while the GP looks after the neurosis service, but we are not very far from this sad state of affairs. The psychiatric services do not have the time, and lack the clinical knowledge and experience, to alter the imbalance. These are inconvenient truths which tend to be concealed, and they have far-reaching educational implications.

Many GPs in their vocational training spend six months attached to a psychiatric team, where they tend to see a highly selected subgroup of patients, unrepresentative of the work of GPs. Only a minute proportion of psychiatrists see anything of general practice during their apprenticeship. At present matters are hardly arranged so that patients are offered the best treatment known to science; nor do doctors get the best education.

Yet the neuroses – so called, for now the terminology is contentious – are so prevalent, distressing and expensive as to constitute an enormous public health problem (Wilkinson 1989*a*), and some fundamental questions remain unanswered. What are the best procedures for explicating, classifying and diagnosing these disorders? Is any conceptualisation valid if it is not based on the multidimensional nature of the phenomena (Cawley, 1983; Jenkins *et al*, 1988)? What are the clinical and other sciences appropriate for treating patients with neurotic disorders in general practice? Does complementary medicine play an important part, and if so why? These and similar questions are gaining recognition internationally

(Wilkinson, 1985). Welcome initiatives are being taken (Strathdee & Williams, 1984; Horder, 1988; Gask & McGrath, 1989), but so far the educational processes, for general practice as well as psychiatry, remain ill-adapted to the realities of these medical and social issues.

Theme 4. Psychiatry does not consist of science alone

Returning to psychiatry as an academic and professional discipline in itself, it seems increasingly important to assert that, by its nature, there is more to it than science. Every year brings significant accretions to the scientific basis of our subject, but much that is important will remain forever outside the range of science. To some this may sound like heresy: but one must remember that the notion that outside science everything is chaos is patently absurd. It is hardly necessary to assert that rigorous thought is possible outside the brains of scientists, and is not ever-present within those powerful networks.

Consider the raw material of the psychiatrist's work: the subjective experiences, thoughts, emotions and behaviour of people in distress and those in close contact with them. The clinical method of psychiatry consists of about seven components – a detailed and many-sided history or biography; the rigorous examination of the patient's predicament, behaviour and mental states and subjective experiences; the physical examination and investigation; the adducing of relevant data and evidence from several sources; the bringing together of all this evidence into a balanced diagnosis and assessment which provide the basis for a sensible and comprehensive programme of management; and the treatment procedures themselves. At the centre is the relationship with the patient, a complex variable as everybody knows. All of this, together with the elaborate business of clinical judgement, forms the stuff of psychiatry. We combine our scientific and practical knowledge, our human and personal attributes, and the fruits of our experience to inform our assessments and management of the individual patient. So ours is an idiographic as well as a nomothetic discipline: that is to say, it recognises and makes much of the uniqueness of the individual as well as the universality of scientific mechanisms. Yet we are sometimes led to suppose that the side of psychiatry which cannot be reduced to science is bogus, and a matter for shame and embarrassment. This will not do. The frontiers between science and non-science are shifting all the time, but I am suggesting that there are irreducible non-science components which are important and neglected. Surely in a psychiatric education they should be recognised, examined, characterised, and discussed. If they are denied, psychiatry is turning its face against a good proportion of its responsibility and its power to help its patients. The psychiatrist of the 21st century will, one hopes, be an informed and disciplined clinical scientist who will not overvalue contemporary science to the extent of practising mere 'scientism'. Two things are required: a clear recognition of the boundaries between science and what is not reachable by science in the background of thought and reasoning which serves psychiatry, and a clearer characterisation of the non-science component.

On this theme there are two further points. Firstly, there is no evidence or logical reason for subsuming the non-science component of psychiatry under the heading of psychoanalysis or psychodynamics. Although these subjects may have something to say about non-science, this is not the place to start. Secondly, the question of the non-science component of our subject is in such need of being recognised and taken seriously that it calls for some initiatives, perhaps even from the Royal College of Psychiatrists. What about a prize to be awarded annually for the best essay or other piece of work on the subject? This would surely evoke some valuable ideas, even some additions to our knowledge of what is amenable to scientific study as well as non-science.

Theme 5. What is the future of psychoanalysis within psychiatry?

My fifth theme introduces a perennially vexed topic. What is the place of psychoanalysis and its derivatives in the modern conspectus of mental illness and its treatment? I think it is right, for several reasons, to separate this question from that of the non-science component of psychiatry. I do not intend to explore the question of whether psychoanalysis is, in whole or in part, a science: it is now clear that this issue is one of massive complexity (see for example Grünbaum's summary of his philosophical critique (1984) together with the peer review by 41 experts, and Grünbaum's response (*Behavioural and Brain Sciences*, 1986)).

Psychoanalysis and the specific theories, models and treatments which have been derived wholly or partially from it relate to a theory or outlook on human existence, development and experience; to a proposed set of aetiological variables – pathogenic or pathoplastic – which affect mental illness; and to a method of treatment. Its proponents, who range widely in their approximations to the central theory, would perhaps be united in saying that no scheme for understanding mental illness and its treatment can be complete without it. Many people who are not themselves psychoanalysts would have sympathy with this view. However, psychoanalysts are inevitably very committed people; and on the other hand there are some committed critics, who emphasise its intangible aspects and the non-refutability of its propositions, and its non-availability for controlled study, and who consequently see the whole body of writings as useless and even pernicious. One of these committed opponents was the late Sir Peter Medawar, a scientist whose contributions to 20th-century biology have earned him a place among the immortals, who went so far as to liken psychoanalysis to a dinosaur or a Zeppelin – a sterile end product, a relic, obsolete and soon to be forgotten, although it "will remain forever one of the saddest and strangest of all landmarks in the history of twentieth century thought" (Medawar, 1982).

Surely he was mistaken. It could be argued that the most striking feature of this aspect of 20th-century thought, and its putative role in understanding and treating mental illness, will prove to be the bleakness of the cold war which even now dominates such an important segment of reasoning in psychiatry. The beginner must be puzzled by the display of pity, scorn, resentment, condescension and superciliousness affected by each side when referring to the other. There is no dialogue, no expressed wish for *rapprochement*, no spirit of compromise, for each faction is totally convinced of the God-given truth of its position. Of course we may try to reassure ourselves by the thoughts that in a sense both sides are right, and most psychiatrists are reasonable men and women who refuse to enter into such extreme degrees of commitment. But even the most reasonable become drawn in, and therefore suspect: it is as if everybody has to be at least a secret follower of one or other camp. It is a stricken area. No dinosaur or Zeppelin ever caused half as much trouble.

This coexistence of viewpoints which are not only disparate but also in active conflict must be conspicuous on the agenda, because the present state of affairs simply will not do for the psychiatrist of the 21st century. He or she should expect to be helped to develop a reasoned and balanced grasp of the matter – one perhaps underpinned by a recognition that there are several kinds of logic, and anyhow it is not logic alone that makes the world go round. Whatever we see as the truth is always on probation. Systems of thought evolve from many different origins, and become entangled with people's feelings, commitment and identities. They are not on that account invalid, although their expression may become unduly dogmatic or distorted.

In the meantime, we can be sure that psychoanalysis will outlive its obituarists. Its continued revered and influential existence is as certain as that of the Greek tragedies or the *Cant*
But its specific beari
tice is a separate ques
and a new look in the h
conceptions than those

Much of the controv
psychoanalysis is qualif
This may not be the m
certainly not the only o
there is scope for explori
For example, Taylor (1
proposals through disti
analytic *theory* and *t*
theory, but drew atte
of such aspects of
forming a concerned
the patient, the cultiva
and countertransference (positive and negative), and the offering of interpretations within this transference relationship. The interpretations do not have to be seen by the therapist as true, but they are directed at helping the patient towards reflection and control. Taylor used these aspects of psychoanalytic technique as the basis of his so-called 'prokaletic' psychotherapy; but of course the principle of separating technique from theory could be applied in other ways, and its usefulness could perhaps be tested.

A second example comes from some work of Hill (1971), who argued for the importance, when considering psychoanalytic theory in relation to general psychiatry, of distinguishing between *mechanism* and *meaning*. At the conventional scientific level, the aetiology and pathogenesis of disorder can be expressed in terms of what is known or supposed about biological, or psychobiological, mechanisms. For the experiencing and sensing individual, meaning is more immediate. By distinguishing carefully between mechanism and meaning, and exploring the latter more diligently than is usual, important questions can be framed in terms of how personal inner meanings can be altered, and whether manipulation of meaning can affect mechanisms. This way of looking at things has common ground with Karl Jaspers' (1963) distinction between *Erklären* and *Verstehen*, explanation and understanding, but it is more open to further thought and empirical research.

A third example is provided by Jackson's (1986) work. Over the years he has repeatedly postulated, and I think demonstrated, that attempts to help in-patients with psychoses and severe personality disorders can be powerfully assisted by recognition, on the part of nurses and doctors, of such variables as transference phenomena, projective identification, introjection – these processes explaining, at least to some extent, the morbid experience of the patient and the bafflement the professional helper tends to experience in the absence of the 'understanding' offered by recourse to this perspective. Hypotheses of this kind are worth testing, and here again is an opportunity for further development and multidisciplinary research.

These examples suggest that there is no shortage of opportunities for scientific inquiry into the place of psychoanalytic principles and techniques in modern psychiatry: if successful, the results should help in establishing what is useful in defined circumstances and, at the same time, tell us something about distinct questions concerning what is science and what is non-science, although not necessarily nonsense.

On a broader scale, the scope for empirical research in dynamic psychotherapy is well illustrated by one further example - the inquiry into the place of family therapy in the treatment of anorexia nervosa by Russell and Dare and their colleagues (Russell *et al*, 1987; Dare *et al*, 1990). This illustrates the need for rather elaborate multidisciplinary work even when the question at issue seems a conceptually simple matter of clinical practice.

It is not appropriate here to review the immense diversity of psychotherapeutic procedures, including those ultimately derived from applied psychoanalysis, those related to social psychology, and those stemming from cognitive and behavioural psychology: the techniques, now well established, which owe so much to the work of psychologists as well as psychiatrists, and provide opportunities for the logical analysis of behaviour, control mechanisms, and techniques of intervention.

An optimist might aver that the outlook for the psychotherapies is bright. If this is correct, much work is required which so far has not even been started. But the psychiatrist of the 21st century may reasonably hope for a clearer charting of the variables and specificities within this broad terrain, and the patient of the 21st century, whatever his/her problems, and however assisted by biomedical advance, may reasonably expect to gain enormously from developments in psychotherapy which are conspicuously possible, if the opportunities for promoting investigative work in the discipline are seized.

Theme 6. Psychiatry in medicine

Psychiatry is a part of medicine, yet we all know there are important ways in which mental illnesses are essentially unlike physical disorders. The symptoms or problems seem very different, although there is much overlap. The psychosocial and experiential dimensions, the supposed aetiology and pathogenesis, the requirements for treatment, the common lack of objective markers, the comparative rarity of pathognomonic features - all seem to underline important contrasts between disorders of the mind and disorders of the body. Consequently, over the last three decades, there has been, in some quarters, a rejection of the so-called 'medical model' in psychiatry, although some people who think this way often resign themselves to an orgy of loose thinking. Psychiatry's necessary and close relationship with other disciplines has evoked suggestions that perhaps mental disorder has little or nothing to do with medicine, so that psychiatry should be demedicalised.

I do not think there is much support for this view nowadays. It is becoming increasingly clear that the distinction between mental and physical disorders, although often striking, is arbitrary and blurred, and unhelpful to the unravelling of disease mechanisms. Psychiatry must continue to be recognised as part of medicine, for the sake of medicine as well as itself - even though it has its own special methods, and its important affiliations with other disciplines.

The psychiatrists of the 21st century will surely have learned to operate more closely in association with medicine than their recent forerunners. The common ground is becoming easier to chart - in terms of neuropsychiatry, liaison psychiatry based on the general hospital, psychiatry in general practice, pharmacology, and the importance of psychiatric audit as a special and extended example of medical audit. Furthermore, the advancement of technologies in general medicine may give the psychiatrist opportunity to contribute towards safeguarding, extending and adapting for special uses the oldest technology of them all - the doctor-patient relationship with all its complexities and vicissitudes.

Within psychiatry, the special field of psychotherapy has sometimes been at pains to demedicalise itself by isolating its work from that of the general hospital and general psychiatric unit, and by emphasising the distance between its methods and those of any other part of medicine or psychiatry. Yet it seems increasingly clear that, if it is to place itself outside the rest of medicine, the special skills of dynamic psychotherapy as a medical specialty will wither and vanish from the scene. Medicine and psychiatry, as well as psychotherapy, would be the losers: both theory and patient care would be sadly deprived.

Theme 7. Management and audit

One cannot avoid making reference to the very modern-sounding issues of management. Like other specialists in clinical medicine, the psychiatrist is in a privileged position in that, however senior he/she becomes, direct care of patients remains possible and indeed the rule. Nurses and social workers by contrast rapidly lose direct contact as they climb the career ladder. They stop being practitioners and become managers. It would be tragic if psychiatric practice was allowed to alter in such a way that consultants became in effect no more than low- or middle-grade managers. Nobody would benefit, least of all the patients. Hard work, ingenuity, and sustained effort will be needed to avoid this state of affairs.

Yet modern management techniques will have to be embraced by the psychiatrist of the future. We can certainly be clear about one thing – the principle of clinical audit has been accepted by the medical profession at large. Everybody agrees it is necessary, in order to assure good practice. And the development and maintenance of accurate and satisfactory audit in psychiatry is going to demand a great deal of work, which to the 21st-century psychiatrist will be unavoidable.

Clinical audit should not be seen as dreary book-keeping. A good system for psychiatric audit would give powerful impetus to the standards of thought and practice in the subject. It would heighten awareness of the really central variables in psychiatric morbidity and its assessment, and in the diversity of treatment procedures. But it is essential that its targets are defined by psychiatrists rather than delegated to managers or computer technologists.

Conclusions

Reverting to Lewis's strictures about time-worn maps, I have briefly surveyed seven broad areas in which psychiatrists of the 21st century may lose their way without charts we do not seem to possess at present. Other areas, perhaps equally important, I have not mentioned, but I have pointed to the need for thought and planning for the day after tomorrow; it behoves us to ask what psychiatry should do now to avoid becoming marginalised. I would like finally to comment on the agents of change. Who is going to lead the way? Before considering this question, let me make two points.

Firstly, by the time a young psychiatrist has emerged from the structured, and even institutionalised, programmes of general and (especially) higher training, he/she is likely to have passed the age by which many scientists of other disciplines have done their best work. Education for good practice is certainly a complex and arduous affair. But we have to ask, and keep asking, whether in this educational process we have the right balance and flexibility. We must always dread the hazards of squandering the best years of people's zest and creativity.

My second point is that, traditionally, the leaders of a profession are its professional body – in our case the Royal College of Psychiatrists – and the senior practitioners and academics, the older consultants and professors – the 'establishment'. Now the Royal College, like all professional bodies, is essentially and necessarily conservative (although not by that token reactionary). It is governed by its Council, elected representatives who collectively represent regional and sectional interests and who are not in a strong position to innovate. Consultants and professors also have their limitations. They have to give close attention to shorter-term developments, conflicts of interest, management and extension of established practice and teaching, and acquisition of funds for research. They have much to occupy them, and in fact they are overstretched perhaps even more than the trainees. Only exceptionally are they able to innovate on a significant scale.

If these two points are accepted, it follows that it may be the trainees (and possibly the junior consultants) who are in the best position to influence the long-term development of the clinical science and profession of psychiatry. I believe we have to look to these ranks to apply the pressure and make the proposals and establish the will for making innovations while conserving all that is good. There is a hard, relentless logic in suggesting that the youngest members of a profession must carry a substantial part of the responsibility for initiating changes to shape its long-term future.

If leads were to be taken by, say, the Collegiate Trainees' Committee of the Royal College of Psychiatrists, and similar groups of young colleagues, it is likely that they would gain impressive support and encouragement from their seniors and others in the health services and even from the wider world. To misuse a phrase borrowed from Aubrey Lewis (1961), the principal agents of cultural advance for the psychiatry of the 21st century may well be among the present trainees.

References

BLAKEMORE, C. (1981) The future of psychiatry in science and society. *Psychological Medicine*, 11, 27–38.

BLEULER, M. (1982) Manfred Bleuler. In *Psychiatrists on Psychiatry* (ed. M. Shepherd), pp. 1–13. Cambridge: Cambridge University Press.

CAWLEY, R. H. (1983) Concepts and classification. In *Handbook of Psychiatry*, Vol. 4. *The Neuroses and Personality Disorders* (eds G. F. M. Russell & L. A. Hersov). Cambridge: Cambridge University Press.

CLARE, A. (1982) Anthony Clare. In *Psychiatrists on Psychiatry* (ed. M. Shepherd), pp. 14–28. Cambridge: Cambridge University Press.

Dare, C., Eisler, I., Russell, G. F. M., *et al* (1990) The clinical and theoretical impact of a controlled trial of family therapy in anorexia nervosa. *Journal of Marital and Family Therapy*, **16**, 39–57.

Fuller, T. E. (1974) *The Death of Psychiatry*. New York: Penguin.

Gask, L. & McGrath, G. (1989) Psychotherapy and general practice. *British Journal of Psychiatry*, **154**, 445–453.

Grünbaum, A. (1984) *The Foundations of Psychoanalysis. A Philosophical Critique*. Berkeley: University of California Press.

—— (1986) *Behavioural and Brain Sciences*, **9**(2), 217–284.

Hill, D. (1971) On the contributions of psychoanalysis to psychiatry: mechanism and meaning. *International Journal of Psychoanalysis*, **52**, 1–10.

Horder, J. (1988) Working with general practitioners. *British Journal of Psychiatry*, **153**, 513–520.

Jackson, M. (1986) A psychoanalytic approach to the assessment of a psychotic patient. *Psychoanalytic Psychotherapy*, **1**, 11–22.

Jaspers, K. (1963) *General Psychopathology* (7th edn) (trans. J. Hoenig & Marion W. Hamilton). Manchester: Manchester University Press.

Jenkins, R., Smeeton, N. & Shepherd, M. (1988) Classification of mental disorder in primary care. *Psychological Medicine* (suppl. 12)

Lewis, A. (1947) The education of psychiatrists. *Lancet*, **ii**, 79–83. (Reprinted in *The State of Psychiatry* (1967) London: Routledge & Kegan Paul.)

—— (1961) *Agents of Cultural Advance*. Oxford: Oxford University Press. (Reprinted in *The State of Psychiatry* (1967) London: Routledge & Kegan Paul.)

Lewis, T. (1934) *Clinical Science*. London: Shaw.

Medawar, P. (1982) Further comments on psychoanalysis. In *Pluto's Republic*, pp. 62–72. Oxford: Oxford University Press.

Pichot, P. (1983) *A Century of Psychiatry*. Basle: Hoffmann-La Roche.

Rawnsley, K. (1984) Psychiatry in jeopardy. *British Journal of Psychiatry*, **145**, 573–578.

Reich, W. (1982) American psycho-ideology. *Bulletin of the Royal College of Psychiatrists*, **6**, 43.

Romano, J. (1982) John Romano. In *Psychiatrists on Psychiatry* (ed. M. Shepherd), pp. 139–151. Cambridge: Cambridge University Press.

Russell, G. F. M. Szmukler, G. C., Dare, C., *et al* (1987) An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, **44**, 1047–1056.

Strathdee, G. & Williams, P. (1984) A survey of psychiatrists in primary care: the silent growth of a new service. *Journal of the Royal College of General Practitioners*, **34**, 615–618.

Taylor, F. K. (1969) Prokaletic measures derived from psychoanalytic techniques. *British Journal of Psychiatry*, **155**, 407–419.

Wilkinson, G. (1985) *Mental Health Practices in Primary Care Settings. An Annotated Bibliography, 1977–1985*. London: Tavistock Publications.

—— (1989*a*) Estimated cost of neurotic disorder in U.K. general practice, 1985. *Psychological Medicine*, **19**, 549–558.

—— (1989*b*) Research report: the general practice research unit at the Institute of Psychiatry. *Psychological Medicine*, **19**, 789–790

R. H. Cawley, BSc, PhD, FRCP, FRCPsych, *Emeritus Professor of Psychological Medicine in the University of London, 24 Earl's Court Square, London SW5 9DN*

British Journal of Psychiatry (1990), 157, 182–189

Further Investigation of the Predictors of Outcome Following First Schizophrenic Episodes

EVE C. JOHNSTONE, J. FIONA MACMILLAN, CHRISTOPHER D. FRITH,
DESMOND K. BENN and TIMOTHY J. CROW

The outcome at two years of patients who were eligible for a study of first schizophrenic episodes was assessed in terms of occupation ($n = 237$) and in terms of number of days spent as an in-patient from the time of first admission ($n = 252$), and was related to social, behavioural, mental state and neurological measures during the initial admission. Poor outcome was in general associated with more social withdrawal, inactivity and abnormal social presentation and with more 'neurological soft signs'. Good occupational outcome in patients with a relatively short pre-treatment duration of illness was associated with the prescription of placebo medication during the follow-up period.

This study is concerned with the outcome of first schizophrenic episodes. The population used in the investigation is that of the Northwick Park study of first episodes of schizophrenia (Johnstone *et al*, 1986). Results concerning outcome in this sample in terms of relapse/readmission within two years of initial admission have already been published (Macmillan *et al*, 1986). Many other studies have examined outcome in schizophrenia and attempted to find predictors (Stephens, 1978; Strauss & Carpenter, 1974; Harrow *et al*, 1986). In comparison with such earlier studies, the present sample of 253 patients is unremarkable in size or duration of follow-up, but it is relatively unusual in that it concerns first-episode patients who were examined, documented and followed up by the same investigators (Macmillan *et al*, 1986). Furthermore, because 120 of the patients were included in a placebo-controlled trial of maintenance neuroleptic medication for the two-year follow-up period (Crow *et al*, 1986), there is an opportunity to assess the effects of neuroleptics upon outcome.

Outcome may be assessed in ways other than of relapse/non-relapse, for example by considering employment and duration of admission to hospital after initial evaluation (Strauss & Carpenter, 1974; Stephens, 1978). Here, outcome is considered in terms of employment at two years and amount of time spent in hospital during the two years following the index admission. The patients were documented at the time of their original admission using the Past History and Socio-Demographic Schedule (PHSD), the Disability Assessment Schedule (DAS), and the Psychological Impairments Rating Schedule (PIRS) (Jablensky *et al*, 1980). In addition, a standardised neurological examination and simple psychological tests were conducted during the initial admission. Predictors of outcome have been sought among these variables.

Method

Between 1 August 1979 and 2 February 1982, 253 patients who fulfilled criteria for a first schizophrenic episode were identified in nine medical centres (Johnstone *et al*, 1986). These patients were followed for two years or until the close of the study, whichever was earlier (Macmillan *et al*, 1986). Outcome was assessed in terms of (a) occupation and (b) number of days within the follow-up period spent as an in-patient.

The occupations of the patients at the time of admission and follow-up were classified in terms of the Registrar General's Classification of Occupations (HMSO, 1980) (this categorises occupations as classes 1–5), which enabled the latter to be classified relative to the former as (a) higher, (b) the same, (c) lower, and (d) no occupation. In the case of mothers of young children not going out to work, full-time child care was considered to be an occupation equivalent to the job which that woman had performed premorbidly. Their employment status was thus assessed by whether their children were being cared for by themselves or by others, even though they themselves were not going out to work. For students and schoolchildren, educational status at follow-up (e.g. whether or not student was at school or university) was classified relative to that before the illness.

The number of days spent as an in-patient from the date of initial admission until either two years had elapsed or the conclusion of the study was counted in each case. The sample was dichotomised at 100 days: those spending less than 100 days were said to have a 'good' outcome and those spending more than 100 days a 'poor' outcome.

Variables from information recorded on a basic information sheet, the PHSD and the DAS (relatives' and nurses' sections) which were used in the calculations, are shown in Tables I, II and III.

The PIRS rates 92 variables, all used in the calculations, which may be classified under three main headings:

TABLE I
Items from a basic information sheet and the Past History and Socio-demographic Schedule

Sex
Ethnic origin
Dates of birth, onset (pre-admission), admission, first trial prescription and discharge
Pre-treatment duration of illness*
Outcome (relapse/readmission)*
Randomised trial treatment (active drugs/placebo)
PSE major class and subclass[1]
Type of onset (acute/subacute/insidious)
Duration of education and educational attainments
Current employment status**, peak of occupational career[2] and most recent occupation[2]
Father's and mother's occupation[2]
Mental illness in father*[3], mother, children, siblings, grandparents, uncles/aunts, cousins, other relatives and spouse
Specified problems in relation to alcohol
Use of illicit drugs (types specified)
Contact with the law in the last 5 years

Significant associations with occupational outcome: $*P<0.01$; $**P<0.001$.

1. Present State Examination (PSE; Wing *et al*, 1974).
2. Registrar General's Classification of Occupations (HMSO, 1960).
3. Interaction between occupational outcome and sex of patient in the case of the father having a psychiatric illness.

(a) activity withdrawal (includes psychic tempo/slowness, attention withdrawal, fatiguability and initiative); (b) social skills (includes communication by facial expression and body language, affect display, conversation skills, and self-presentation); and (c) global impressions of patient's personality.

'Neurological soft signs' have previously been studied in relation to psychiatric disease in general (Hertzig & Birch, 1966, 1968; Mosher *et al*, 1971; Rochford *et al*, 1970) and to schizophrenia in particular (Quitkin *et al*, 1976; Woods *et al*, 1986). The term 'neurological soft signs' refers to any neurological lesion which does not localise the site of a putative central nervous system (CNS) lesion. The causation of these signs is unclear, and they do not lead to inference of pathologies such as tumours or vascular lesions. They are non-life-threatening and anatomically non-specific but have been found to correlate with low IQ (Mosher *et al*, 1971; Quitkin *et al*, 1976); it has been suggested that they may result from CNS damage and may be caused by intrauterine or perinatal injury or anoxia (Quitkin *et al*, 1976). Examination for 'soft signs' in the present study consisted of most of the items used by Quitkin *et al* (1976), but also included assessments of tone and spontaneous involuntary movement disorder. The tests used are summarised in Table IV. Simple psychological tests (Withers & Hinton, 1971), Peabody Picture Vocabulary Test (Dunn, 1965) and the Digit Symbol Substitution test from the WAIS (Wechsler, 1958) were also conducted on the same occasion.

The Disturbed Behaviour Rating (DBR; Johnstone *et al*, 1986) was devised by the authors to record in a systematic manner the sometimes very disturbed behaviour which the

TABLE II
Items rated on Disability Assessment Schedule (relatives section) by relatives and by interviewer

Items *Reported functions rated by relative (parent/spouse/other) acting as informant*[1]	*Modifying factors assessed by interviewer*
Self-care	Regular activity to increase skills/knowledge
Underactivity	Special interests or activities
Slowness	Above average ability or assets
Social withdrawal*	Privileged circumstances or environment
Participation in household activities	Confiding relationship other than parent or spouse*
Affective relationship with spouse	Disadvantaged current environment
Sexual relationship with spouse	Physical, neurological, or sensory handicap
Interest in and care of children	Other unfavourable characteristics
Heterosexual role	Key figure in home
Friction in interpersonal relationship outside household	Emotional involvement of key figure
Occupational role	Controls and demands by key figure
Interest in getting a job	Rejection of patient by key figure
Behaviour in crisis or emergency	Patients access to privacy in home
	Outside support sought
	Outside support received
	Ease of obtaining job if patient had no mental illness
	Subjective overall assessment of patient's social adjustment*
	Subjective impression of characteristics of key figure (includes self-insight)

Significant association with occupational outcome: $*P<0.01$.

1. Each function rated: (a) as it has been in the past month; and (b) as compared with situation one year previously.

TABLE III
Items rated on Disability Assessment Schedule (nurses' section)

Nurses' descriptions of behaviour and activities	*Nurses' opinions regarding patients' abilities and requirements*
Slowness of movement	Ability to do useful work^{2}
Underactivity	Suitability to possess matches
Overactivity	Suitability to visit friends at weekends^{*1}
Conversation	Suitability to go out with patients of opposite sex
Social withdrawal^{*2}	Suitability to possess scissors or razor blades
Leisure interests	Ability to look after earned money^{*1,2}
Irrelevant or incomprehensible talk^{*2}	Ability to work outside while living in hospital
Posturing and mannerisms	Ability to be discharged to suitable accommodation and job
Threatening and violent behaviour	Requirements for a locked room or ward
Personal appearance	Reasons for constant supervision if needed
Behaviour at meal-times	
Housekeeping activities on ward	
Occupational therapy	
Other social therapy	
Visits to patient	
Patient's visits home	
Parole status	

Significant association with outcome in terms of occupation (1) or in-patient days (2): $^{*}P<0.01$.

TABLE IV
Tests used for examinations of neurological soft signs

Tone – all 4 limbs separately	Stereognosis
Handedness for writing	Nystagmus
Finger–thumb opposition (precision grip) right & left	Blepharospasm
	Reading
Apraxia (6 postures each rated separately)	Writing
	Glabellar response
Pupillary movements	Eye movements
Voluntary facial movements	Draw an arrow
Loss of normal accessory movements	Gait on walking
	Heel to toe (6 steps)
Abnormal accessory movements	
Movement disorders of face (lips and mouth, jaw, tongue, trunk, upper & lower limbs)	

patients demonstrated before admission. It records: (a) behaviour potentially threatening to the life of the patient; (b) behaviour potentially threatening to the lives of others; (c) disturbed or inappropriate sexual behaviour; (d) behaviour damaging to property; (e) bizarre or inappropriate behaviour which cannot be classified in any of the above terms. Each item is rated as (i) absent, (ii) on at least one occasion, (iii) repeatedly during the month prior to admission, or (iv) repeatedly for more than one month prior to admission.

The items of the PHSD, DAS and PIRS, the neurological soft signs, the psychological test results and the items of the DBR were all related to outcome in terms of occupation and in-patient days.

In terms of occupation, the patients were divided into four groups (better, the same, worse, no occupation – see above) and these were subsequently collapsed for most calculations into two groups, good (better and the same) and poor (worse and no occupation). The two outcome groups were compared (with sex as an additional factor) by applying χ^2 statistics to the frequencies in the different categories of the variable being considered. The two outcome groups in terms of in-patient days (<100 days or >100 days) were compared in the same way. Because of the very large numbers of comparisons being examined, only those significant at the 1% level or less in this initial calculation are considered. To take account of the possibility that some of the variables giving significant results may be related, a subsequent multivariate analysis was conducted (stepwise logistic regression). As it was found that age of onset and pre-treatment duration of illness were significant determinants of number of in-patient days ($P<0.001$ and $P<0.02$ respectively) these determinants together with sex were used in the multivariate analysis. The multivariate analysis could not be applied to all parts of the data because complete information was not available on some of the schedules, particularly the DAS (relatives section).

Results

Outcome in terms of occupation

A total of 253 patients were eligible for the initial study (Johnstone *et al*, 1986). In this analysis, all available follow-up information was used including that on the four patients who died and those who entered judicial custody, and that which became available to the authors after the follow-up period. There were 16 patients on whom no information in terms of occupational status was available. The outcome in terms of occupation in the remaining 237 patients is shown in Table V (subdivided by ethnic origin and sex). There was no significant relationship between outcome in terms of occupation and age, sex or ethnic origin. For the remainder of the calculations, outcome in occupational terms is divided into good and poor (see earlier).

Basic information and PHSD

As shown in Table I, occupational outcome related significantly to relapse/readmission ($P<0.01$ – no relapse, better outcome), pre-treatment duration of illness ($P<0.01$ – shorter duration, better outcome) and in the trial cases, to the nature of the experimental treatment ($P<0.01$, better outcome in patients on placebo). It had, however, been shown that there was a trend for patients who were to go on active drug to have a longer pre-treatment duration of illness (Crow *et al*, 1986), and therefore, in order to take account of this difference, the effect of treatment upon outcome was analysed separately for those with a pre-treatment duration of illness of (a) more than one

TABLE V
Outcome in terms of occupation, subdivided by race and sex

Ethnic race	*Occupation at follow-up relative to that at admission*							
	Better		*Same*		*Worse*		*Unemployed*	
	male (n)	*female* (n)	*male* (n)	*female* (n)	*male* (n)	*female* (n)	*male* (n)	*female* (n)
Caucasian	5	1	33	31	4	4	46	33
Afro-Caribbean	0	1	10	2	2	1	14	8
Afro	0	0	2	0	1	2	3	3
Asian	0	0	5	6	1	2	5	3
Chinese	0	0	0	0	0	0	0	1
Mixed	0	0	3	1	0	0	3	1

year and (b) less than one year. For the former patients there was no significant effect of treatment upon outcome, but for the latter ones there was a significant effect such that outcome was better ($P<0.05$) in patients on placebo. Apart from this effect of experimental treatment, the only significant direct relationship was between the unemployed status and poor occupational outcome two years later ($P<0.001$). There was a significant interaction between outcome and sex such that psychiatric illness in the father was associated with relatively good outcome in male patients and relatively poor outcome in female patients. The multivariate analysis confirmed the significant effect of pre-treatment duration of illness.

DAS (relatives' section)

Table II shows that poor outcome in occupational terms was significantly related to social withdrawal ($P<0.01$), worsening social withdrawal in the past year ($P<0.01$), absence of a confiding relationship ($P<0.01$), and patients' overall social adjustment assessed as poor ($P<0.01$). Underactivity and worsening underactivity in the past year were each separately and significantly at the 1% level associated with a poor occupational outcome in men and a good occupational outcome in women. It was not possible to perform multivariate analysis on this section of the DAS because of incomplete information.

DAS (nurses' section)

None of the described behaviour and activities (Table III first column) were significantly associated with occupational outcome. Some significant relationships were established with nurses' opinions (Table III, second column) in that unsuitability to visit friends ($P<0.01$) or to possess money ($P<0.01$) were associated with poor outcome, and these results were confirmed by multivariate analysis. Unsuitability to be discharged was associated with poor outcome for men only ($P<0.01$).

PIRS

Only lack of 'special assets' ($P<0.001$) was significantly associated with poor occupational outcome. These included 'intelligence', which was separately considered and in the group as a whole showed no relationship with outcome, and 'good looks' which showed a weak relationship ($P<0.05$). However, the asset most often described was 'charm of manner' (patients described as pleasant, socially graceful, socially well preserved, charming, etc.), and it would appear that 'charm', so useful a quality in the well, retains its value in the psychotic. The strong relationship between poor outcome and lack of special assets was confirmed by multivariate analysis ($P<0.001$). Relationships between poor outcome and abnormal quietness and limited use of gesture also emerged ($P<0.02$).

Neurological soft signs and psychological tests

These tests could only be performed on 137 patients. The remainder were unable or unwilling to co-operate. None of the items was significantly associated with occupational outcome. There was one highly significant association and this related to whether or not it was possible to perform tests: the occupational outcome of those on whom the tests could not be done was worse ($P<0.001$). The reasons for non-performance concerned the patients' mental state. Sometimes the patient was hostile and suspicious, and for this reason would not do the tests, but more commonly patients could not comply with instructions because of inattention, distractibility and disorganisation of behaviour.

DBR

Poor occupational outcome was significantly related to behaviour threatening to the lives of others ($P<0.01$) and to bizarre or inappropriate behaviour which could not be classified under any specific heading ($P<0.01$). These relationships were confirmed by multivariate analysis.

Outcome in terms of in-patient days

The information regarding in-patient days was obtained from the patient and/or an informant. It was checked with the hospital to which the patient had been admitted and cross-checked with local hospitals and with the other hospitals in the study. Information was obtained on 252 patients, the remaining patient and her relatives refused to discuss the issue with the investigators.

Outcome in terms of in-patient days (good <100 days, poor >100 days), as might be expected, was related to

P, 25, 428

relapse/readmission ($P<0.01$) so that those who relapsed had more in-patient days than those who had not.

Basic information and PHSD

As shown in Table I, there were no direct significant relationships between any of these items and outcome in terms of in-patient days.

DAS (relatives' section)

Table II shows that there were no relationships significant at less than 1% between any items of this schedule and outcome in terms of in-patient days.

DAS (nurses' section)

Two described behaviour and activities were associated with worse outcome in terms of in-patient days (Table III) – more social withdrawal ($P<0.01$), and the presence of irrelevant or incomprehensible talk ($P<0.01$). Worse outcome was also associated with the nurses' opinions regarding the patient's inability to do useful work ($P<0.01$) and to look after money ($P<0.01$). These two nurses' opinion items were confirmed with multivariate analysis, which also showed a trend to an association between poor outcome and social withdrawal ($P<0.09$) and revealed an association with posturing and mannerisms.

PIRS

Poor outcome (> 100 days) was significantly associated with reduction of facial movements ($P<0.01$), reduction of quantity of facial expression ($P<0.01$), more facial mannerisms ($P<0.01$) and a more abnormal overall impression in terms of facial communication ($P<0.01$), more severely blunted affect ($P<0.01$), flat conversation ($P<0.001$) and disturbance of listener feedback ($P<0.01$). In contrast to the relationships with occupational outcome, lack of 'special assets' was only weakly associated with this outcome. The associations with reduction of facial movements, quantity of facial expression, blunted affect, flat conversation and lack of special assets were confirmed by multivariate analysis.

Neurological soft signs and psychological tests

Psychological test performance was not associated with outcome in terms of in-patient days, but a number of neurological soft signs were. These included abnormal precision grip on the left ($P<0.01$), abnormal glabellar response ($P<0.01$), abnormal gait ($P<0.01$), and loss of normal accessory movements on walking ($P<0.001$). There was a highly significant interaction ($P<0.0001$) between outcome, movement disorder of the tongue, and sex, such that for men, those who had fewer than 100 in-patient days had a slightly greater frequency of abnormal tongue movement (30%) than those who had more than 100 in-patient days (23%) with abnormal response; while for women, the former group had a substantially lesser frequency (17%) than the latter one (65%). Multivariate analysis confirmed the association between poor outcome and abnormal precision grip, glabellar response, loss of accessory movements and movement disorder of the lips and tongue, and using this technique, an association with poor performance on picture vocabulary emerged.

These results have to be considered against the background of the drug treatment that these patients were having. Neurological examination was conducted during the index admission once the patient was accustomed to the investigators and was able to co-operate with testing. As the patients were almost always started on neuroleptics shortly after admission, with three exceptions, all patients were having neuroleptic drugs at the time of the neurological examination. More than minimal neuroleptic medication before admission was an exclusion criterion for the project (Johnstone *et al*, 1986), but when the neurological examinations were conducted at a mean of six weeks after admission, the patients who were included had had variable (and changing) amounts of a range of neuroleptics and anticholinergic drugs.

DBR

There were no significant associations between any of the items of the DBR and outcome in terms of in-patient days.

Discussion

The findings should be considered with caution. Firstly, a large number of comparisons are made so that some significant results are likely to arise on the basis of chance alone. Secondly, there is no control group in this study; some factors associated with particular outcomes may have little to do with schizophrenia, but reflect a general association between two sets of circumstances. For example, unemployed status at the time of admission is associated with poor occupational outcome two years later. It is obvious that this is likely to be true of other patient groups and indeed of non-patient samples. Similar considerations may apply to other less obvious associations.

Another factor that should be borne in mind is the stage of the illness. It has previously been shown that the effects upon outcome of pre-morbid social functioning are substantially less in schizophrenic patients at an early stage in their illness. It is also suggested (Harrow *et al*, 1986) that some of the previously reported positive findings regarding pre-morbid functioning may have been influenced by patients' characteristics at hospital admission that are a function of the severity of an already developed illness rather than of pre-morbid adjustment. This should not be as powerful an effect in the present study as it has been thought to be in more heterogeneous samples (Strauss & Carpenter, 1974; Carpenter *et al*, 1978). However, although these patients were all first-episode cases, the length of time between the estimated onset and admission to hospital did vary and satisfactory arrangements for admission were often difficult to make (Johnstone *et al*, 1986). The possibility therefore remains that some features which are associated with poor outcome are due to already established illness. The design of the present study minimises but does not eliminate this possibility.

Associations with outcome in terms of occupation

The association of poor outcome in occupational terms and relapse/readmission is predictable, and since greater pre-treatment duration of illness is so strongly predictive of relapse/readmission (Crow *et al*, 1986), it is not surprising that it is also associated with poor occupational outcome. As far as experimental treatment was concerned, active treatment was associated with a significantly smaller chance of relapse (Crow *et al*, 1986). In spite of this, a significant relationship was shown between better outcome in occupational terms and placebo rather than active drugs. The possibility that placebo medication might be associated with benefits of this kind was raised by the previous finding that of 13 of the 253 trial entrants and eligibles who made achievements of an educational, occupational or social nature, nine were on placebo medication (Macmillan *et al*, 1986). The present finding may be considered as a stronger indication of the possible social cost of maintenance neuroleptic medication.

The direct associations between poor occupational outcome and items of the DAS (relatives' section) concern social withdrawal and isolation in some sense. The relationships between such factors and poor outcome in schizophrenia are well established. The possibility that shyness and friendlessness would have an association with employment difficulties in the population as a whole is clearly raised; in the absence of a control group, however, no specific association with schizophrenia can be assumed. Similar considerations are relevant to the sex differences in determinants of occupational outcome among the DAS (relatives' section) variables. Slowness and underactivity are associated at the 1% level with a poor occupational outcome in men but a good one in women. The nature of these symptoms may differ between the sexes; for example, it could be that while they might indicate schizophrenia with a poor prognosis in men they could indicate schizophrenia with an affective element and hence a better prognosis in women. On the other hand, it is possible that these associations are not illness-related but indicate that a lack of activity and speed cause difficulties in employment for men but are valuable for women in their jobs.

With regard to the DAS (nurses' section), the nurses' opinions that the patients would not be able to behave responsibly were associated with poor occupational outcome. These variables are likely to be independent and the associations indicate the practical value of the subjective impressions of experienced staff. With regard to the PIRS, the association between lack of special assets (principally social charm) and poor outcome is striking ($P<0.001$), and the unexpected element about this is not the direction of the association but the fact that the impression that a substantial proportion of schizophrenic patients retain their social charm and presence received a degree of validation two years later.

The lack of association between the individual neurological signs and occupational outcome was disappointing. It is likely that the non-performance of the neurological examination was a measure of severity and as such its association with poor outcome is understandable. The associations with poor outcome of the items of the DBR (behaviour threatening to the lives of others and bizarre or inappropriate behaviour) are further examples where the association could exist whether or not schizophrenia was an issue. Clearly, a known history of behaviour of this kind would be likely to be associated with employment difficulties.

Outcome in terms of in-patient days

Outcome in terms of occupation at two-year follow-up is likely to be reasonably independent of the ratings made during the index admission, but this is clearly not true of outcome in terms of in-patient days. This measure consists of the initial admission plus any subsequent periods of in-patient care. The duration of the initial admission (and possibly the threshold for subsequent admissions) is likely to vary from hospital to hospital and between one consultant and another. Because of the small numbers of patients at some hospitals (Crow *et al*, 1986) and the very large number of collaborating consultants, it is difficult to assess these effects. It is perhaps more relevant that the duration of in-patient care is likely to have been influenced by the opinions and observations of the staff and perhaps also of the relatives included in the assessments used in these calculations. The DAS ratings (nurses' section) are the least independent of outcome in terms of in-patient days, but the observations in general point to the association between withdrawal and lack of activity and poor outcome. The nurses' opinions are bound to have influenced the duration of admission and this is likely to be relevant to the fact that the items of the PIRS which were associated with more in-patient days concerned severity of behavioural disturbance in terms of withdrawal and social presentation.

The large number of significant associations between poor outcome in terms of in-patient days and neurological soft signs is a striking finding and contrasts with the lack of associations with occupational outcome. As mentioned, it is not possible to determine the extent to which issues related to neuroleptic medication have affected this result. Although in some previous studies such as that of Quitkin *et al* (1976), most patients were withdrawn from neuroleptic drugs for 10 days before assessment, in others such as that by Woods *et al* (1986) this was not so. Ideally, patients who have never had medication should be studied, but the findings of the present study in which a substantial proportion of first-episode cases were not able to co-operate with neurological assessment even though they were receiving neuroleptic medication underline the impracticality of such an investigation. In the study of Woods *et al* (1986), the examining neurologists made a clinical decision as to whether or not the abnormalities found were related to medication. Validation of such judgements is difficult and no similar attempt was made in the present study. Our view is that in the absence of a non-schizophrenic control group, we cannot know to what extent the findings reflect the illness, the treatment or are unrelated.

The fact remains, however, that the presence of a number of signs is significantly associated with outcome. This may be because patients who were more severely ill and therefore likely to spend more time over the next two years as in-patients had received more medication. It may be because patients with a poor prognosis have neurological soft signs irrespective of medication or because such patients develop them in response to medication to a greater degree than those who do not require so much in-patient care. It is not possible to differentiate between these possibilities on the basis of the available evidence, but similar findings of an association between the presence of neurological soft signs and schizophrenia of poor outcome (and with poor pre-morbid social functioning) led Quitkin *et al* (1976) to conclude that there was likely to be an organic basis for this disorder. The most significant finding in relation to neurological soft signs is the interaction between movement disorder of the tongue, outcome and sex. The fact that some drug-induced extrapyramidal effects have been noted to be commoner in females (Ayd, 1961; Marsden *et al*, 1986) may be relevant to this result.

Conclusions

In summary, poor outcome is in general associated with social withdrawal, inactivity and poor social presentation, and with more soft neurological signs. Many previous studies (e.g. Gittleman-Klein & Klein, 1969; Rosen *et al*, 1971; Bromet *et al*, 1974; Harrow *et al*, 1986) have shown an association between poor outcome in various terms and pre-illness social withdrawal, and it has been previously suggested that soft neurological signs are associated with poor progress in schizophrenia (Quitkin *et al*, 1986; Torrey *et al*, 1980). The other principal finding of the study is the significant association in patients with a relatively short pre-treatment duration of illness between better outcome in occupational terms and placebo medication, in spite of the significant associations between relapse and poor outcome, and relapse and placebo medication. This is a new finding because placebo-controlled follow-up studies of first episodes of schizophrenia are very rare (Crow *et al*, 1986). It suggests the disquieting conclusion that the benefits of active neuroleptics in reducing relapse may exact a price in occupational terms. This emphasises the need to find more effective means of predicting which patients derive benefit from maintenance neuroleptics and which do not. Although our earlier analysis established a significant advantage for active medication in reducing relapse (Crow *et al*, 1986) in that approximately 60% of patients on placebo and 40% of patients on active medication relapsed in the first two years after an initial schizophrenic episode, 40% of patients did *not* relapse although they were not on active medication. The present results emphasise the importance of finding a way to determine the patients whose illness will run this course.

Acknowledgements

This investigation was carried out under the auspices of the Ethical Committees of Northwick Park Hospital and the other hospitals concerned. Thanks are due to the patients, their relatives and the nursing staff for their co-operation and to Mrs Asmita Soni for typing the manuscript.

References

Ayd, F. J. (1960) A survey of drug induced extra pyramidal reactions. *Journal of the American Medical Association*, 175, 1054–1060.

Bromet, E., Harrow, M. & Kasl, S. (1974) Premorbid functioning and outcome in schizophrenics and non-schizophrenics. *Archives of General Psychiatry*, 30, 203–207.

Carpenter, W. T., Bartko, J. J., Strauss, J. S., *et al* (1978) Signs and symptoms as predictors of outcome: a report from the International Pilot Study of Schizophrenia. *American Journal of Psychiatry*, 135, 940–944.

Crow, T. J., Macmillan, J. F., Johnson, A. L., *et al* (1986) The Northwick Park study of first episodes of schizophrenia. II. A randomised controlled trial of prophylactic neuroleptic treatment. *British Journal of Psychiatry*, **148**, 120–127.

Dunn, L. M. (1965) *Expanded Manual for the Peabody Picture Vocabulary Test*. Minneapolis: Minneapolis American Guidance Service.

Gittleman-Klein, R. & Klein, D. F. (1969) Premorbid asocial adjustment and prognosis in schizophrenia. *Journal of Psychiatric Research*, **7**, 35–53.

Harrow, M., Westermeyer, L. F., Silverstein, M., *et al* (1986) Predictors of outcome in schizophrenia: the process-reactive dimension. *Schizophrenia Bulletin*, **12**, 195–206.

Hertzig, M. & Birch, H. (1966) Neurologic organization in psychiatrically disturbed adolescent girls. *Archives of General Psychiatry*, **15**, 590–598.

—— & —— (1968) Neurologic organization in psychiatrically disturbed adolescents: a comparative consideration of sex differences. *Archives of General Psychiatry*, **19**, 528–537.

Her Majesty's Stationery Office (1980) *Classification of Occupations of the Registrar General*. London: HMSO.

Jablensky, A., Schwartz, R. & Tomov, T. (1980) World Health Organization collaborative study on impairments and disabilities associated with the schizophrenic disorders. A preliminary communication: objectives and methods. *Acta Psychiatrica Scandinavica*, **62** (suppl.), 285.

Johnstone, E. C., Crow, T. J., Johnson, A. L., *et al* (1986) The Northwick Park study of first episodes of schizophrenia. I. *British Journal of Psychiatry*, **148**, 115–120.

Macmillan, J. F., Gold, A., Crow, T. J., *et al* (1986) The Northwick Park study of first episodes of schizophrenia. III. *British Journal of Psychiatry*, **148**, 128–133.

Marsden, C. D., Mindham, R. H. S. & Mackay, A. V. P. (1986) Extrapyramidal movement disorders produced by antipsychotic drugs. In *The Pharmacology and Treatment of Schizophrenia* (eds P. B. Bradley & S. R. Hirsch), pp. 340–402. Oxford: Oxford Medical Publications.

Mosher, L., Pollin, W. & Stabenau, J. (1971) Identical twins discordant for schizophrenia: neurologic findings. *Archives of General Psychiatry*, **24**, 422–430.

Quitkin, F., Rifkin, A. & Klein, D. F. (1976) Neurological soft signs in schizophrenia and character disorders. *Archives of General Psychiatry*, **33**, 845–853.

Rochford, J. M., Detre, T., Tucker, G. J., *et al* (1970) Neurophysiological impairments in functional psychiatric diseases *Archives of General Psychiatry*, **22**, 114–119.

Rosen, B., Klein, D. F. & Gittleman-Klein, R. (1971) The prediction of rehospitalisation: the relationship between age of first psychiatric treatment contact, mental states and premarked asocial adjustment. *Journal of Nervous and Mental Disease*, **152**, 17–22.

Stephens, L. H. (1978) Long-term prognosis and follow-up in schizophrenia. *Schizophrenia Bulletin*, **4**, 25–47.

Strauss, J. S. & Carpenter, W. T. (1974) The prediction of outcome in schizophrenia. II. Relationships between predictors and outcome variables. *Archives of General Psychiatry*, 31, 37–42.

Torrey, E. F. (1980) Neurological abnormalities in schizophrenic patients. *Biological Psychiatry*, **15**, 381–338.

Wechsler, D. (1958) *The Measurement and Appraisal of Adult Intelligence*. Baltimore: Williams & Wilkins.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

Withers, E. & Hinton, J. (1971) Three forms of the clinical tests of the sensorium and their reliability. *British Journal of Psychiatry*, **119**, 1–8.

Woods, B. T., Kinney, D. K. & Yurgelun-Todd, D. (1986) Neurologic abnormalities in schizophrenic patients and their families. *Archives of General Psychiatry*, **43**, 657–663.

*Eve C. Johnstone, MD, FRCP, FRCPsych, *formerly Member of Scientific Staff and Honorary Consultant Psychiatrist, Northwick Park Hospital and Clinical Research Centre, Watford Road, Harrow, Middlesex, HA1 3UJ, now Professor of Psychiatry, University Department of Psychiatry, The Kennedy Tower, Royal Edinburgh Hospital, Edinburgh EH10 5HF*; J. Fiona Macmillan, MD, MRCPsych, *Senior Lecturer in Psychiatry, University of Birmingham (formerly Senior Registrar, Northwick Park Hospital and Clinical Research Centre)*; Christopher D. Frith, MA, Dip Psych, PhD, *Member of Scientific Staff, Northwick Park Hospital and Clinical Research Centre*; Desmond K. Benn, MRCPsych, *formerly Registrar in Psychiatry, Northwick Park Hospital and Clinical Research Centre, now Senior Registrar in Psychiatry, Northern Regional Health Authority*; Timothy J. Crow, PhD, FRCP, FRCPsych, *Head of Division and Honorary Consultant Psychiatrist, Northwick Park Hospital and Clinical Research Centre*

*Correspondence

British Journal of Psychiatry (1990), 157, 190–196

Alcoholism: A Follow-up Study of Participants in an Alcohol Treatment Programme

G. K. SHAW, SETA WALLER, SINE McDOUGALL, JENNIE MacGARVIE and GRAHAM DUNN

One hundred and twelve alcoholic patients treated by an intensive one-month residential programme were followed up for one year. As a group, they were socially disadvantaged and highly dependent on alcohol. Outcome of treatment was assessed at six months and one year following discharge by multiple measures which included assessments of drinking behaviour, measurements of social stability, neuroticism and self-esteem, and self-ratings of satisfaction with important aspects of day-to-day living. During the first six months following treatment, 37% were abstinent or drinking in controlled fashion; during the second six months, 53% achieved this status. Improvement in drinking status was positively related to improvements in all other outcome variables.

Over the past decade or so it has been increasingly recognised that the majority of people with alcohol-related problems neither require nor particularly benefit from relatively lengthy periods of in-patient treatment (Miller & Hester, 1986*a*,*b*; Edwards *et al*, 1987; Edwards, 1988). There are, therefore, legitimate doubts about the role of Alcohol Treatment Unit (ATU) programmes and the type of patients likely to benefit from them. Reports of outcome studies from British ATUs have been scanty and this report, in essence an audit of some of the work of our unit, contributes to the body of knowledge on outcome expectation in a defined subgroup of alcoholics.

Most British ATUs prefer lesser interventions whenever possible. Thus, of 140 consecutive new patients seen in one of our ATU's out-patient clinics in 1985, only 41 (29%) were ever admitted to the unit throughout the period ending in December 1987. Most of those patients were admitted for brief periods of detoxification because of severe chemical dependence on alcohol and some were admitted because they were disabled by significant physical illness, significant brain damage or by severe neurotic or psychotic symptoms. Only seven patients (5%) were admitted to our full-scale treatment programme. Simpler interventions involving advice, counselling or support on an out-patient basis were preferred for the majority.

Patients are selected for this treatment programme by a multidisciplinary team who consider all referrals from unit staff or outside agencies. In general, those selected have failed to benefit from less intensive therapies conducted over a lengthy time period and are not disabled by psychosis or significant brain damage.

Assessment, by multiple measures, of outcome at six months and one year following the completion of the programme is reported for 112 participants.

Treatment programmes of different ATUs vary, and thus some description is necessary. Firstly, there is no requirement that participants should be committed to an abstinence goal. The programme includes group therapy, counselling, training in social skills and relaxation techniques, as well as educational material. Structured and unstructured group sessions offer patients an opportunity to consider their life circumstances, their lifestyle and their relationship with alcohol. They are asked to reflect upon the benefits and disadvantages consequent upon their drinking and to decide whether they wish to make changes in their lifestyle and their use of alcohol and, if so, what kind of help would most usefully enable them to do so.

The treatment team do not subscribe to a narrow medical view of alcoholism as an illness and accept that the disposition to drink excessively is widely determined. The programme is, however, conducted in a hospital setting and staff are health care workers. Thus, participants may formulate their problems within a medical frame.

The programme lasts for four weeks and 6–10 patients are admitted *en bloc* to participate in each one. On completion, all patients are offered follow-up appointments as appropriate.

Method

All 112 alcoholic patients consecutively admitted to a four-week intensive treatment programme between April 1984 and January 1986 were included in this study. Computerised psychometric test scores for this group were midway between those of a group of normal controls and those of an average group of alcoholic patients admitted to this ATU (Acker *et al*, 1984). There were 92 men and 20 women. Of the 112 patients, 104 were followed up for six months and 91 for one year.

Procedure at intake

All patients were interviewed at intake by an experienced sociologist who collected basic sociodemographic data relating to age, marital status, socioeconomic status according to the Registrar General's Classification of Occupations (1980), level of education (age at which person left full-time education), and living circumstances.

Further data were obtained through more detailed questioning. Where necessary, specific probes were used and the time of six months before admission was taken as a point of reference. The following variables were investigated in this way.

(1) 'Employment' – the number of days of employment in the previous six months.
(2) 'Criminal involvement' – the number of alcohol-related and non-alcohol-related offences.
(3) 'Admission to hospital' – the number of in-patient days in general hospitals, psychiatric hospitals or alcohol units.
(4) 'Number of visits to medical and psychiatric facilities' – the total visits to a general hospital, psychiatric hospital, alcohol clinic or general practitioner.
(5) 'Total number of visits to social agencies' – this includes visits to Alcoholics Anonymous (AA), an alcohol counselling service or similar agency, and religious institutions.
(6) 'Admissions to a detoxification unit' – the number of separate admissions for detoxification.
(7) 'Social stability' – this scale is derived from Straus & Bacon (1951). One point was awarded for each of the following (maximum score = 4):
    (a) married or cohabiting in the previous six months;
    (b) employed for at least five of the previous six months;
    (c) no criminal offences in previous six months;
    (d) living in own home or rented accommodation (as opposed to lodgings, hostels, or being of no fixed abode) in previous six months.
(8) 'Drinking behaviour' – this includes;
    (a) number of units consumed on a typical heavy drinking day (unit = ½ pint of beer, 1 glass of wine, or 1 single measure of spirit) assessed through a detailed reconstruction of a recent heavy drinking day;
    (b) number of units consumed on a typical controlled drinking day, assessed as above;
    (c) 'pattern of drinking' – categories include frequent and infrequent drinking bouts (frequency and duration recorded) with periods of abstinence or controlled drinking, as well as continuous alcoholic drinking;
    (d) total number of days abstinent in the previous six months;
    (e) longest period of abstinence (number of weeks);
    (f) total number of years of heavy drinking.
(9) 'Physical complications and consequences of drinking' – the incidence of withdrawal symptoms was graded as mild or severe; delirium tremens, fits, alcoholic amnesias, relief drinking and loss of control was noted (scale 0–10).
(10) 'Social complications of drinking' – the number of times subject had borrowed or stolen money for drink, pawned or sold possessions, lost a job or had trouble with friends, neighbours or police through drinking was recorded (scale 0–infinity, range 0–109).
(11) 'Neuroticism' – assessed by self-administered Crown–Crisp Experimental Index (Crown & Crisp, 1979) (scale 0–96; upper limits of normal males 22.5, females 29).
(12) 'Self-esteem' – assessed by the self-administered Litman Scale (Litman *et al*, 1983) (scale 18–90, *lower* scores indicate *higher* levels of self-esteem).
(13) 'Dependence on alcohol' – assessed by the Severity of Alcohol Dependence Questionnaire (SADQ; Stockwell *et al*, 1983) (scale 0–60, scores greater than 30 indicate severe dependence).
(14) 'Satisfaction with life situations' – this is a new self-assessment measure. Patients are asked to rate their level of satisfaction with the following life situations on a five-point scale (very satisfactory (1) to very unsatisfactory (5); scale 7–35, *lower* scores indicate *greater* satisfaction): (a) Relationships; (b) Employment; (c) Accommodation; (d) Use of day; (e) Use of leisure; (f) Physical health; (g) Mental health.

Procedure at six-month and one-year follow-up

With the exception of basic sociodemographic data, all measures and scales were repeated at six months and one year. This yielded two study periods: intake to six months and six months to one year. Also at six months and one year, the 'pattern of drinking' variable included two further categories: abstinent and controlled drinking.

At monthly intervals throughout the study, patients were contacted whenever possible to improve the accuracy of the data collected at formal follow-up stages.

All statistical analyses were carried out using the statistical package SAS (SAS, 1985).

Results

Drop-outs

Eight patients dropped out before the six-month follow-up. One patient had died of brain haemorrhage, three patients living in temporary accommodation had moved on and a further four patients who were placed in 'dry houses' left soon after admission. Another 13 patients dropped out between the six-month and one-year follow-ups. Two of these had gone abroad, the remaining 11 had changed address and could not subsequently be contacted.

Characteristics of the sample

The mean age of the sample was 40.6 years (s.e.m. 0.75, range 19–61), with an average of 9.11 (s.e.m. 0.51) years of problem drinking. They scored highly on measures of alcohol dependency (SADQ 31.17 (s.e.m. 1.25)) and

TABLE I

Means (s.e.m.) of social, psychological, drinking and self-rated variables at intake, six months and one year

Variable	*Intake* (n = *104*)	*Six months* (n = *104*)	*One year* (n = *91*)
Social stability	2.00 (0.11)	2.22 (0.12)*	2.46 (0.12)
Self-esteem	50.38 (1.49)	45.57 (1.70)*	42.48 (1.36)
Neuroticism	51.85 (1.45)	38.98 (2.12)**	33.27 (2.34)
Satisfaction with life situations (total scores)	24.06 (0.68)	19.97 (0.79)**	18.8 (0.81)
Days in hospital	16.73 (1.81)	8.99 (1.45)**	5.00 (1.13)
Physical complications of drinking	2.62 (0.11)	1.50 (0.15)**	2.35 (0.33)
Social complications of drinking	10.44 (1.66)	5.95 (1.29)*	5.87 (1.83)
Number of days of abstinence	77.96 (5.62)	130.87 (6.10)**	139.40 (6.68)
Longest period of abstinence (in weeks)	5.53 (0.55)	14.71 (1.003)**	16.39 (1.15)
Number of days off work	121.18 (6.87)	115.14 (7.95)	104.20 (9.21)

*$P<0.01$, **$P<0.001$

neuroticism (Crown–Crisp Index 52.63 (s.e.m. 1.39)), and were of average intelligence on the National Adult Reading Test (NART; Nelson, 1982), scoring a mean of 28.4 (s.e.m. 1.13). As a group, their current social class was lower than their highest ever social class, and 70% were unemployed. Thirty-seven per cent were married or cohabiting, the remainder being single, separated, widowed or divorced. Twenty per cent were of no fixed abode.

Changes in sociodemographic and drinking variables

Table I lists various sociodemographic, drinking and self-rated variables at intake, six months and one year. For statistical purposes the means at intake are based on the 104 patients followed up for six months. With the exception of the number of days off work, the improvement between the mean at intake and at six-month follow-up was always statistically significant. Since the means at one year are better than those at six months, it follows that the differences here are also statistically significant. In the case of days off work, the change from intake to the one-year follow-up stage just fails to achieve statistical significance ($P<0.06$).

There were progressive group improvements in social stability, self-esteem and satisfaction with all life circumstances assessed as well as significant reductions in neuroticism. The mean number of days admitted to hospital progressively fell as did the physical and social complications of drinking experienced. Days of abstinence and longest period of abstinence increased. Days off work were reduced although not to a statistically significant degree.

Changes in drinking pattern

At the six-monthly and one-yearly follow-up stages, patients were categorised as 'abstinent', 'controlled', 'improved' or 'unchanged/worse' drinkers. 'Abstinent drinkers' were those patients who did not drink at all over the relevant six-month period. 'Controlled drinkers' were those whose maximum consumption level did not exceed eight units of alcohol a week or four units on any drinking occasion. 'Improved drinkers' were so categorised if there had been a shift in their drinking pattern to a less pathological drinking style and if this was associated with a marked reduction in units of alcohol drunk per time period. 'Unchanged drinkers' continued to drink as much or more than at intake.

By comparison with the six months before entry to the study, the 'controlled drinkers' had reduced their total alcohol consumption by 98% over both six-month study periods. In simple terms, as a group they now drank in the course of a year what formerly they had drunk in the course of a week.

The group categorised as 'improved drinkers' at the six-month follow-up stage had decreased their consumption by 86% over that time period. Those so categorised at the one-year follow-up stage had decreased their consumption by 82% between the six-month and one-year follow-up compared with consumption over the six months before intake. As a group they now drank in the course of a week what formerly they would have drunk in a day.

These drinking categories are related to other outcome variables in Tables II and III. It is clear that the four groups defined in terms of their drinking status at six months also differ in outcome measured by psychological and social variables. The groups are, however, differently composed with respect to these variables at intake and, in the case of social stability, neuroticism, satisfaction with life situations and physical complications the differences are statistically significant ($P<0.05$) as assessed by a non-parametric test equivalent to a one-way analysis of variance, the Kruskall–Wallis test (NPAR1WAY procedure in SAS (SAS, 1985)). The possibility that differences at six months merely reflected differences at intake was examined by investigating the statistical significance of improvement scores (the difference between scores at six months and scores at intake). Since the four groups differ in their variability as well as their mean levels of improvement this was also assessed by the Kruskall–Wallis test. In all cases the groups were found to be different ($P<0.001$). A closer examination of the data in Tables II and III reveals that the improvement in the abstinent, controlled and improved drinking groups is essentially the same while the unchanged group always deteriorated.

TABLE II

Means (s.e.m.) of social and psychological variables of patients grouped by drinking status at six-month and one-year follow-up

Drinking status	*Social stability*		*Neuroticism*		*Self-esteem*		*Satisfaction with life situation*	
At six months (n = *104)*	*intake*	*6 months*	*intake*	*6 months*	*intake*	*6 months*	*intake*	*6 months*
Abstinent (*n* = 30)	2.27 (0.23)	2.73 (0.22)	46.70 (2.79)	20.50 (2.43)	48.66 (2.28)	35.66 (1.70)	23.13 (0.63)	15.03 (0.62)
Controlled (*n* = 8)	2.87 (0.51)	3.37 (0.26)	44.62 (7.16)	25.75 (4.49)	43.75 (7.05)	37.50 (6.74)	19.75 (1.99)	13.87 (0.85)
Improved (*n* = 25)	1.92 (0.26)	2.29 (0.22)	54.71 (2.17)	31.46 (2.45)	50.83 (2.81)	38.75 (2.71)	24.37 (0.83)	17.58 (0.65)
Unchanged/worse (*n* = 41)	1.71 (0.15)	1.58 (0.17)	55.28 (2.23)	59.00 (2.30)	52.61 (2.57)	58.09 (2.48)	25.40 (0.61)	26.12 (0.64)
At one year (n = *91)*	*intake*	*1 year*	*intake*	*1 year*	*intake*	*1 year*	*intake*	*1 year*
Abstinent (*n* = 42)	2.33 (0.20)	2.98 (0.14)	46.71 (2.30)	18.52 (1.83)	48.57 (1.91)	34.09 (1.08)	23.14 (0.60)	14.52 (0.46)
Controlled (*n* = 6)	3.17 (0.54)	3.50 (0.50)	45.33 (8.20)	18.17 (3.71)	43.33 (8.81)	30.83 (2.05)	19.33 (2.17)	14.33 (1.22)
Improved (*n* = 16)	1.81 (0.26)	2.37 (0.18)	59.19 (2.93)	34.25 (4.29)	53.12 (4.05)	43.50 (2.01)	25.19 (1.06)	18.94 (1.09)
Unchanged/worse (*n* = 27)	1.67 (0.17)	1.48 (0.20)	54.22 (2.65)	59.00 (2.93)	50.37 (2.94)	57.52 (1.78)	24.92 (0.73)	26.44 (0.73)

Significance of differences between improvement scores in different drinking status groups for all parameters $P < 0.001$ (Kruskall–Wallis test).

TABLE III

Means (s.e.m.) of variables relating to admission to hospital and complications of drinking of patients grouped by drinking status at six-month and one-year follow-up

Drinking status	*Out-patient visits*		*Days in hospital*		*Physical complications*		*Social complications*	
At six months (n = *104)*	*intake*	*6 months*	*intake*	*6 months*	*intake*	*6 months*	*intake*	*6 months*
Abstinent (*n* = 30)	4.93 (0.62)	2.57 (0.73)	16.23 (4.20)	0.23 (0.23)	2.23 (0.19)	–	8.26 (3.39)	–
Controlled (*n* = 8)	8.37 (1.72)	1.87 (0.81)	8.87 (4.29)	1.75 (1.75)	2.00 (0.50)	–	7.00 (4.27)	0.12 (0.12)
Improved (*n* = 25)	7.29 (0.77)	3.67 (0.77)	22.00 (4.64)	4.46 (1.63)	2.83 (0.23)	1.25 (0.20)	13.29 (3.72)	1.50 (0.54)
Unchanged/worse (*n* = 41)	6.40 (0.84)	7.74 (0.86)	15.57 (1.82)	19.21 (2.79)	2.90 (0.16)	3.02 (0.14)	11.02 (2.46)	13.85 (2.79)
At one year (n = *91)*	*intake*	*1 year*	*intake*	*1 year*	*intake*	*1 year*	*intake*	*1 year*
Abstinent (*n* = 42)	5.67 (0.58)	2.50 (0.65)	16.09 (3.21)	–	2.33 (0.18)	–	9.92 (2.38)	–
Controlled (*n* = 6)	5.50 (1.02)	–	5.33 (2.65)	–	2.16 (0.54)	–	5.50 (4.41)	–
Improved (*n* = 16)	7.94 (1.00)	2.81 (0.71)	20.81 (4.17)	3.94 (1.42)	2.75 (0.28)	3.12 (0.68)	10.93 (4.18)	1.12 (0.46)
Unchanged/worse (*n* = 27)	6.74 (1.21)	5.96 (0.86)	19.63 (3.97)	14,52 (3.03)	2.92 (0.20)	6.07 (0.45)	13.26 (3.62)	19.15 (5.45)

Significance of differences between improvement scores in different drinking status groups, on all parameters $P < 0.001$ (Kruskall–Wallis test), with the exception of, at the one-year stage, out-patient visits $P < 0.01$ and days in hospital $P < 0.05$.

TABLE IV

Classification of outcome in terms of drinking status after six months and one year

Drinking status	*Six months (n = 104)*	*One year (n = 91)*
Abstinent	30 (29%)	42 (46.2%)
Controlled	8 (8%)	6 (6.6%)
Improved	25 (24%)	16 (17.6%)
Unchanged/worse	41 (39%)	27 (29.6%)

The groups at one year are differently constituted but the pattern of results is similar. The abstinent, controlled and improved drinking groups improve in terms of all variables while the unchanged group usually deteriorate. In all cases, the differences between the groups are statistically significant as assessed by the Kruskall–Wallis test.

Table IV lists outcome by drinking status at six-month and one-year follow-up. At six months, 30 patients were abstinent and by the year end the situation had improved since a further 12 patients had been recruited to this category.

Table V charts the changes in drinking status which occurred between the six-month and one-year follow-up stages. The italic entries indicate those who maintained the same status at each follow-up stage. Data to the left of these italic figures indicate those who improved between follow-up stages, figures to the right those who deteriorated. Thus, for example, between the follow-up stages, 20 patients improved, 3 deteriorated and 13 were lost to follow-up. For the purpose of analysis all 13 missing patients were allocated to the unchanged group. Thus, 20 patients were regarded as improved and eight as deteriorating. Statistical testing of marginal homogeneity (using the CATMOD procedure in SAS (SAS, 1985)) finds the trend towards improvement between the follow-up stages to be statistically significant ($P < 0.02$).

Discussion

The assessment of outcome of treatment in alcoholic patients is beset by difficulties. The strategy of this study has been to compare the social circumstances and drinking status over the six months before treatment with the situation over two consecutive six-monthly post-treatment periods.

Data collected when patients are under research scrutiny are likely to be more accurate than pre-study assessments. For example, at the six-month follow-up stage the data (incomplete) that had been collected by monthly contact with the patients and records of clinical team contacts were available which both improved the accuracy of quantitative data and increased the confidence with which patients could be allocated to drinking-status categories. However, the method of case selection described earlier meant that programme participants had usually been known to the unit for months or years. Their in-patient and out-patient case records were, therefore, available to the research team when collecting retrospective data.

There is no control group or comparison with alternative treatment methods. Thus, it is possible that the sample might have benefited to a similar or greater extent from alternative approaches. The group were selected, however, on the grounds that they had failed to benefit to date from less intensive interventions which included counselling, out-patient attendance and usually one or more admissions for brief periods of detoxification. The time scale of one year is short but the consensus from the literature is that outcome at one year relates closely to outcome in the longer term.

Participation in this treatment programme was followed whenever possible by out-patient follow-up, and those of no fixed abode were placed in appropriate accommodation. Post-discharge experiences of these and many other kinds may have been as important as the index treatment in the determination of outcome, a dilemma common to all outcome studies.

The diversity of patients subsumed under the umbrella term 'alcoholism' is such that it is difficult to feel confident when comparing results of one

TABLE V

Changes in drinking status between the six-month and one-year follow-up stages

Status at six months	*Status at one year*				
	Abstinent	*Controlled*	*Improved*	*Unchanged*	*(Missing)*
Abstinent ($n = 30$)	*28*	–	1	–	(1)
Controlled ($n = 8$)	3	*5*	–	–	(–)
Improved ($n = 25$)	8	1	*10*	2	(4)
Unchanged/worse ($n = 41$)	3	–	5	*25*	(8)
Missing ($n = 8$)	–	–	–	–	*(8)*
Totals	42	6	16	27	(21)

study with another that they are based on similar populations. Thus, we have outlined as clearly as possible the characteristics of our sample: they were socially disadvantaged; two-thirds were single, separated, widowed or divorced; two-thirds were unemployed; and 40% were either of no fixed abode or lived alone.

Of those who participated in our treatment programme and were available to follow-up, 37% were abstinent or drinking in controlled fashion at six months and one year respectively. These gradings are based on interview assessments and self-report. They are to some extent, however, 'validated' by the level of agreement with other outcome variables shown in Tables II and III.

Self-report in the field of alcoholism is not highly regarded, yet the weight of the research evidence reviewed by Midanik (1988) suggests that self-report agrees well with reports from collaterals, particularly when the specific amount of alcohol taken is not the prime focus of attention. Differences when they occur are not consistently in the direction of under-reporting by alcoholics.

Midanik (1988) also concludes that self-reported data, such as we have used, on drinking arrests, days in hospital and use of other agencies relate closely to data collected from official records and that discrepancies are in the direction of under-reporting from inadequately maintained official records.

The outcome of this treatment group compares favourably with the usual findings in 'no treatment' studies. Thus, Kendell & Staton (1966) reported 24% of untreated alcoholics either abstinent or drinking in controlled fashion at follow-up, Imber *et al* (1976) 11–19% depending on the time interval, and Vaillant (1980) 17% on the basis of pooled studies which included the Kendell & Staton and Imber *et al* findings.

These results also compare favourably with 'advice only' studies, although the characteristics of the samples are very different. Thus, Chick *et al* (1988) reported 17% of patients either abstinent or drinking without problems in 96 attenders at alcoholic out-patients and Edwards *et al* (1977), in a group of married, relatively stable alcoholics, reported 'slight or no' drinking problem in approximately one-third of patients.

There are two points of interest. Firstly, the results of the second six months of the study indicate significant improvements by comparison with the first six months. Secondly, those patient groups who successfully curtailed their drinking achieved considerable improvements on all social and psychological parameters examined.

There are important implications for health care economics. Those who achieved abstinent or controlled drinking status required no days in hospital throughout the subsequent year – a reduction of 16 days and seven days respectively from the six-monthly period before treatment. Those whose drinking was rated as improved had a reduced mean number of days in hospital from an estimated 21 days per six months before treatment to eight days throughout the subsequent year. Those whose drinking remained unchanged required 34 days' admission to hospital during the year following treatment. The pattern of out-patient visits is similar.

Although simpler interventions are preferred for the majority of patients presenting to alcohol clinics, there is support in the literature for the view that the most seriously disadvantaged and severely dependent alcoholics may require more intensive treatments, sometimes involving residential placement. Indeed, on the basis of an extensive literature review, Miller & Hester (1986*b*) conclude that "more severe and less socially stable alcoholics seem to fare better in in-patient (or more intensive) treatment". The Edwards *et al* (1977) 'treatment versus advice' study of a relatively stable, married alcoholic population has been most influential in promoting simple interventions, but the same research group, in a further analysis of their data, point out that their most highly dependent group, Gamma alcoholics, "appeared to benefit from relatively intensive treatment" (Orford *et al*, 1976).

The present study suggests that a significant proportion of a disadvantaged group of alcoholics benefits from an intensive residential treatment programme both in terms of drinking status and of social and psychological well-being. Nonetheless, 30–40% of the participants did not improve. There are indications from a closer examination of the complete study data (report in preparation) that psychological impairment was the most important predictor of outcome, highlighting the need to look for alternative treatment approaches in the brain-damaged subgroup.

A practical strategy for the management of patients with alcohol-related problems would be to deal with the majority by non-intensive interventions on an out-patient basis; the highly chemically dependent may require detoxification on an in-patient, an ambulatory or a domiciliary basis, and the socially disadvantaged and highly dependent may require more intensive interventions often involving residential placement. Outcome in this disadvantaged group is not universally poor, but those who suffer significant cognitive impairment are likely to do badly and make heavy demands on health-care resources. As yet, the knowledge and skills to deal effectively with such patients is lacking, implying an urgent need to study their problems and to develop strategies for their treatment and rehabilitation.

Acknowledgements

This study was funded by a DHSS Grant no. 10146. Grateful thanks are due to Elmdene Unit treatment staff who run the treatment programme reported on and to Mrs J. Johns for invaluable secretarial assistance.

References

Acker, C., Acker, W. & Shaw, G. K. (1984) Assessment of cognitive function in alcoholics by computer: a control study. *Alcohol and Alcoholism*, 19, 223–233.

Chick, J., Ritson, B. Connaughton, J., *et al* (1983). Advice versus extended treatment for alcoholism: a controlled study. *British Journal of Addiction*, **83**, 159–170.

Crown, S. & Crisp, A. H. (1979) *Crown–Crisp Experiential Index*. Kent, UK: Hodder & Stoughton.

Edwards, G., Orford, J., Egert, S., *et al* (1977) Alcoholism: a controlled trial of "treatment" and "advice". *Journal of Studies on Alcohol*, **38**, 1004–1030.

Edwards, G (1988) Which treatments work for drinking problems? *British Medical Journal* (editorial), **296**, 4–5.

Imber, S., Schultz, E., Funderburk, F., *et al* (1976) The fate of the untreated alcoholic. *Journal of Nervous and Mental Disease*, **162**, 238–247.

Kendall, R. E. & Staton, M. C. (1966) The fate of untreated alcoholics. *Quarterly Journal of Studies on Alcohol*, **27**, 30–41

Litman, G., Stapleton, J., Oppenheim, A. N., *et al* (1983) An instrument for measuring coping behaviour in hospitalised alcoholics: implications for relapse, prevention, treatment. *British Journal of Addiction*, **78**, 269–276.

Midanik, L. T. (1988) Validity of self-reported alcohol use: a literature review and assessment. *British Journal of Addiction*, **83**, 1019–1029.

Miller, W. R. & Hester, R. K. (1986*a*) The effectiveness of alcoholism treatment – what research reveals. In *Treating Addictive Behaviours* (eds W. R. Miller & N Heather, pp. 121–174). New York: Plenum.

Miller, W. R. & Hester, R. K. (1986*b*). Inpatient alcoholism treatment: who benefits? *American Psychologist*, **41**, 794–805.

Nelson, H. E. (1982) National Adult Reading Test. Windsor: NFER Nelson.

Orford, J., Oppenheimer, E. & Edwards, G. (1976) Abstinence or control: the outcome for excessive drinkers two years after consultation. *Behaviour Research and Therapy*, **14**, 409–418.

Registrar General (1980) Classification of Occupations. Surrey: Office of Population Census.

SAS (1985) *Statistics* (version 5). Cary, NC: SAS Institute.

Stockwell, T., Murphy, D. & Hodgson, R. (1983) The severity of alcohol dependence questionnaire: its use, reliability and validity. *British Journal of Addiction*, **78**, 145–155.

Straus, R. & Bacon, S. D. (1951) Alcoholism and social stability: a study of occupational integration on 2023 male clinic patients. *Quarterly Journal of Studies on Alcohol*, **12**, 231–260.

Vaillant, G. E. (1980) The doctor's dilemma. In *Alcoholism Treatment in Transition* (eds G. Edwards & M. Grant), pp. 13–31. London: Croom Helm.

*G. K. Shaw, FRCPsych, DPM, *Consultant Psychiatrist, Bexley Hospital, Old Bexley Lane, Bexley, Kent*; Seta Waller, MA, *Research Sociologist, Bexley Hospital, Old Bexley Lane, Bexley, Kent*; Sine McDougall, MA, *Research Psychologist, Bexley Hospital, Old Bexley Lane, Bexley, Kent*; Jennie MacGarvie, BSc, *Statistician, Institute of Psychiatry, London SE5*; Graham Dunn, MA, MSc, PhD, *Senior Lecturer in Statistics, Institute of Psychiatry, London SE5*

*Correspondence

British Journal of Psychiatry (1990), 157, 197–207

The Case-Control Study in Psychiatry

GLYN LEWIS and ANTHONY J. PELOSI

Case-control studies involve 'cases' being compared to 'controls' with respect to 'exposures', possible aetiological (or associated) factors. Associations between a disease and an exposure can be explained by chance, reverse causality, confounding and biases or, lastly, by causality. However, confounders as well as information and selection biases can be adjusted for at the design (or analysis) stage of the study. The strength of an association can be measured by means of relative risk, calculated indirectly using the odds ratio. Well conducted control studies should produce accurate estimates of relative risks in many psychiatric investigations.

"In a case-control study, individuals with a particular condition or disease (the *cases*) are selected for comparison with a series of individuals in whom the condition or disease is absent (the *controls*). Cases and controls are compared with respect to existing or past attributes or exposures thought to be relevant to the development of the condition or disease under study" (Schlesselman, 1982).

Case-control studies thus estimate the strength of association between a disease and a factor of interest and are widely used as a quick, efficient, and relatively inexpensive means of exploring possible aetiological associations. Mantel & Haenszel (1959) observed that the method is "adapted to the limited resources of an individual investigator and places a premium on the formulation of hypotheses for testing, rather than on facilities for data collection".

The refinement of this methodology has been one of the major advances in epidemiological research over the past three decades. Many of the technical developments have been by epidemiologists interested in the aetiology of cancer who have done much to remove the disreputable image of the 'quick and dirty' case-control study that provided unreliable results requiring confirmation with other study designs. The case-control study now has a central respected place in aetiological research.

Its application to mental disorders will also involve those particular problems which bedevil the psychiatric researcher. However, psychiatry is only a special case within medicine and the similarities between our problems and those of our medical colleagues are more striking than the differences. Where possible we shall attempt to discuss the particular problems of psychiatry and how they have been or could be overcome.

This paper is intended to help those planning studies as well as those who want to understand more about interpreting the results of scientific papers. Also, professional bodies in all areas of medicine, including psychiatry, are stressing the need for a more thorough knowledge of epidemiology and experience of research.

There is insufficient space to explain fully all the topics covered here, but reference will be made to more detailed accounts. General problems of study design, interpretation and confounding will be outlined, before discussing those difficulties which raise particular problems in case-control studies: selection bias and information bias.

Nomenclature

Exposure

Epidemiologists tend to use 'cases' to refer to cases of the disease or condition and 'exposure' to describe the possible aetiological (or associated) factor of interest, although in many circumstances the latter term is clearly inappropriate. 'Exposures' will be broadly defined and will not be restricted to a history of past contact with, for instance, a noxious chemical, infection or social adversity. A current physiological characteristic or an ongoing or long past contact with a possible causal factor will all be called 'exposures'. Case-control studies have thus been used to study a range of psychiatric disorders and exposures, such as the association between schizophrenia and computerised tomography (CT) scan parameters (e.g. Smith *et al*, 1988) and obstetric complications (e.g. Lewis & Murray, 1987), risk factors for tardive dyskinesia (e.g. Mukherjee *et al*, 1986) and depression and corticosteroid metabolism (e.g. Carroll *et al*, 1981). Geneticists who study the association of a disorder with an increased familial risk are also using a case-control design (e.g. Coryell & Tsuang, 1982).

This illustrates that epidemiology crosses the ideological boundaries of psychiatry, and its methods are as essential for those interested in biological factors as for social psychiatrists. There is nothing more 'biological', for instance, than the link between smoking and lung cancer, and this association was discovered using a case-control study (Doll & Hill, 1952).

Types of study

The nature of case-control studies will be clarified by comparing them with other designs. In the past, the terms 'prospective' and 'retrospective' have been used to describe cohort and case-control studies but these terms are sometimes used to refer to the way in which the data was collected rather than the logical basis of the study. Hopefully the following classification of types of study will avoid this ambiguity.

(1) Clinical description

Describing a series of cases of a disease is still a useful and important research activity. However, the association between a condition and an exposure is rarely so strong that a clinical description provides enough evidence for it. For less clear-cut associations, a comparison group is needed in order to estimate the prevalence of the history of exposure in the general population.

(2) Case-control studies

The case-control study can therefore be seen as an extension of the traditional clinical description of cases. The proportion of cases giving a history of exposure is compared with that in the controls. The control group gives an estimate of exposed proportion in the population from which the cases were drawn. The researcher therefore usually starts by selecting a source of cases and then chooses an appropriate control group. This is often the most difficult step in designing a case-control study (discussed later). Although 'control' is not very appropriate to describe members of the comparison group, alternatives have not gained widespread acceptance.

(3) Cross-sectional surveys

A population is screened for both the disease and the exposure, allowing the association to be studied. In cross-sectional surveys the cases are identified by means of a population-based survey, and cases can be compared with the non-diseased subjects in the study. Therefore, cross-sectional surveys overcome the important problem of selection bias (see below). However, it has the disadvantage that for uncommon conditions, large numbers of subjects have to be screened in order to detect enough cases.

(4) Cohort or longitudinal studies

The researcher selects two groups without the disease but who differ in terms of their exposure to the possible aetiological factor. The subjects are followed up and the incidence of disease in the two groups compared. For rare diseases this will obviously involve following up very many subjects, but with the more common minor psychiatric disorders, this kind of study can be feasible.

(5) Clinical trials and other experimental designs

It is rarely ethical to study aetiology with an experimental design, although treatment response can sometimes provide clues to the cause of disorder. The key feature which differentiates this type of study is that the investigator randomly allocates subjects (or occasionally clinics or factories) to two or more groups which are treated in different ways. This design randomly allocates all confounding variables, including those of which the researcher is unaware. Confounding can still occur, but does so by chance, and the probability of this is statistically estimated. The main problems in interpretation arise from drop-out from treatment, bias in measurement and uncertainty about generalising the findings. The latter problem and the expense of clinical trials have led some authors to advocate using case-control designs to study treatment (Smith *et al*, 1984).

Choosing a study design in uncommon conditions

Case-control studies have a particular advantage in investigating uncommon illnesses. For instance, the incidence of schizophrenia (less than 20 per 100 000 per year) means that over 500 000 recruits would be required to expect 100 new adult cases in a year's follow-up. For this reason case-control studies, which start with identified cases of schizophrenia rather than healthy subjects, are much more feasible than cohort studies (but see Andreassen *et al* (1987) for an example of the latter).

Studying and interpreting associations

Case-control studies, cross-sectional surveys and cohort studies can measure associations between a disease and exposure and by such studies researchers attempt to study disease aetiology. An associated exposure may be referred to as a 'risk factor', which can sometimes, but not invariably, imply causality. There are well documented difficulties in inferring cause from an observed association and guidelines to assist these inferences (Hill, 1965). There are four main explanations to be considered when an association is observed.

(1) Chance

An association can arise by chance, and statistical tests of significance estimate the probability of this occurring (the '*P*' value). This means that a result significant at the 5% level will, on average, be found once in every twenty studies even if no true association exists. When this occurs it is known as a type one error and is a particular problem if repeated significance tests are performed within a study.

Less commonly recognised is the opposite error, that a real association is missed because a study is too small and the variation correspondingly large; the study therefore lacks statistical power. Such type two errors are a particular problem in biological studies in psychiatry because the investigations are expensive and sample sizes are therefore very small.

Calculating confidence intervals provides more information than a single '*P*' value (Gardner & Altman, 1986) because it indicates the statistical precision of an estimate as well as the probability of the result occurring by chance.

(2) Reverse causality

This refers to the observation that the disease may cause the exposure. This is often a problem in case-control studies because data on exposure are usually collected retrospectively. For instance marital discord may be associated with depression because depression leads to marital problems. Likewise, low social class, which is associated with schizophrenia, is widely held to result from the illness rather than be a cause of it (Goldberg & Morrison, 1963). In case-control studies therefore, the timing of the exposure and onset of disease is of importance. Similarly, in life-events research much attention is paid to 'independent' life events as opposed to those which may be influenced by the illness under study (Brown & Harris, 1978). In comparison, cohort studies usually start by identifying exposed subjects who do not, at that stage, have the disease, and so reverse causality is less of a problem.

Reverse causality can be an explanation of interest. Some authors have suggested that the association between results of the dexamethasone suppression test and depression has aetiological significance. In contrast, Mullen *et al* (1986) have suggested that changes in corticosteroid metabolism result from the anorexia and insomnia of depression rather than being a possible cause.

(3) Confounding and bias

These are the most important of the possible interpretations of an association. Adjusting for confounding in design and analysis is one of the major preoccupations of medical research. A confounder is an independent risk factor (or protective factor) for the disease at each level of the exposure and is also associated with the exposure. It is not therefore a variable on the causal pathway. A confounding factor can lead to a spurious association or can eliminate a real association between exposure and disease (see below).

There are many potential sources of bias in scientific investigations and the most important of these in case-control studies, selection and information bias are discussed later.

(4) Causality

The last explanation for an association is that the exposure does increase the risk of disease, that it is a cause or on the causal pathway.

Inferring a cause from an association

The main hindrance in making the leap from association to aetiology concerns the problem of confounding. Geoffrey Rose's aphorism that "one cannot account for the confounding factor one does not know about" is a useful reminder when designing studies.

Random allocation of individuals to experimental groups reduces the problem of confounding but people are not randomly allocated to possible causes of disease. Observational studies (case-control, cohort studies and cross-sectional surveys) remain the only way to study environmental causes, and so the problem of confounding is introduced. In attempting to interpret an association it is often helpful to ask "'what are the determinants of exposure in this group, why were some exposed others not?'. In other words, 'How does this study situation differ from a randomised trial?'" (Rose, personal communication).

The final conclusion depends upon judgement, argument and the uses to which the conclusion will be put. In studies which bear directly upon important areas of social policy, one would expect more convincing evidence to be provided by the researcher. Laboratory work provides plausible final pathways for causes, thus supplementing epidemiological studies that indicate the importance of causal factors in the population at risk.

Incident or prevalent cases

In studying aetiological associations it is recommended that, where possible, new incident cases of disease are selected. If prevalent cases are studied there will be an over-representation of subjects with a poor prognosis who have had the condition for a long period. Therefore an observed association may indicate risk factors for a poor prognosis rather than for the onset of disease.

Size of effect v. statistical significance

The usual method of measuring the strength of an association in aetiological studies is by means of relative risk, i.e. the increased incidence rate of disease in the exposed group expressed as a multiple of the rate in the unexposed group. Cohort studies can estimate the relative incidence rates directly, but in case-control studies this can only be done indirectly using the odds ratio, a simple calculation illustrated in the Appendix.

The use of relative risk gives a measure of the strength of an association and not its statistical significance. This distinction is especially important because the strength of an association is a pointer to aetiological importance (Hill, 1965). For instance, cigarette smoking increases the risk of lung cancer about 10 times, and increases the risk of ischaemic heart disease by 2.5 times. In general, most epidemiologists regard a relative risk above 1.5 or 2 as a risk factor of aetiological interest. Estimates of the relative risk of association between recent life events and depression are around five. Winter birth appears to increase the risk of schizophrenia by less than 1.1 (Eaton, 1985).

Precision: statistical power and study size

The statistical power gives the probability that a type two error will *not* occur. This is more likely to occur when the estimate produced by a study is imprecise. The main method of increasing statistical power is to increase the sample size. In small studies which produce statistically insignificant results a type two error should be considered.

The power of a study depends upon four factors: (1) the strength of the expected association; (2) the prevalence of exposure; (3) the significance level, usually taken as 5%; (4) the sample size. For example, a case-control study looking for a relative risk of two at the 5% level where the prevalence of exposure is 15% has a power of 80% when there are 200 cases and 200 controls.

Unless the researcher is expecting a very strong association between the disease and exposure, a case-control study needs to be quite large to have sufficient statistical power. Although the case-control approach usually needs less resources than cohort studies or surveys, by current standards within psychiatry, statistically powerful case-control studies would still be considered large investigations.

An example of a case-control study

Craig & Brown (1984) used the case-control method to study the possible importance of life events in the aetiology of painful gastrointestinal (GI) conditions. They identified those suffering from 'functional' illness in a GI clinic and compared them with a group from the same clinic who had organic GI disorder and with another group of healthy people from the local community. Table I has been constructed from just some of their results; an environmental stress is defined as a severe life event or major difficulty in a 38-week period preceding the interview.

The efficiency and relative cheapness of the design is clear. If Craig & Brown had designed a cross-sectional community survey to test their hypothesis they would have had to interview many thousands of people before a sufficient number with gastrointestinal disorders would be identified.

In asking whether these life events are of causal significance in functional bowel symptoms the other possible explanatory factors (chance, reverse causality and confounding and bias) must first be considered. The difference in the cases and controls may have arisen by chance but this is most unlikely, as indicated by the usual tests of statistical significance. The 95% confidence limits for the odds ratio are from 2.8 to 13.1. Functional bowel illness could conceivably lead to a greater number of life events (i.e. reverse causality), perhaps mediated by their effects on work performance, relationships, etc. However, the assessment plays close attention to

TABLE I

Life events and painful gastrointestinal (GI) disorder (results adapted from Craig & Brown (1983))

	Functional GI disorder	*Organic GI disorder*
Environmental stress	53	26
No environmental stress	14	42
		Odds ratio = 6.1

timing and independence. Sex and social class, for example, are possible confounders and a number of possible biases have to be considered. Selection and referral biases appear to be the most important. For example, are those with functional bowel symptoms more likely to be referred for hospital treatment if they have recently had life stresses and does this apply to the same extent to the hospital controls?

Confounding

In any observational research the true association between disease and hypothesised exposure can be distorted by confounding variables. Confounders can either lead to a spurious association or eliminate the association between a real risk factor and the disease. Confounding therefore results in a biased estimate of relative risk. If it can be measured then the analysis can be adjusted to take the confounder into account. This makes consideration of confounding essential at the design stage of a study.

A simple illustration is provided by the observation that more women have Alzheimer's dementia than men. This results from two other associations. Firstly, there is a relationship between age and Alzheimer's disease (that it becomes commoner with age) and secondly, women are more likely to live longer than men. Thus age is said to confound the relationship between sex and Alzheimer's disease. Age is a common confounder and needs to be taken into account in almost any study.

Why and how confounding can occur is difficult to explain and is best illustrated using a hypothetical numerical example (Table II) which has been deliberately designed with unrealistically large associations. Suppose a study is attempting to measure the association (if any) between life events and schizophrenia. In the raw data, illustrated in Table II*a*, there is no association, with an odds ratio of one. However, schizophrenics are less likely to have children at home, and if we further suppose that life events are commoner in households that have children, the presence of children in the home confounds the relationship between schizophrenia and life events. When the results are stratified by the presence or absence or children (Table II*b*), there now appears to be a strong association previously masked by the confounder with an overall odds ratio of around five.

A confounder is associated with the disease at each level of the exposure. This is illustrated in Table II*c*, as the data are now stratified by the exposure, life events. The odds ratios now illustrate the association between the confounder (the presence of children) and the disease. The odds ratio of 0.05 in this hypothetical example indicates that schizophrenics are about 20 times *less* likely to have children at home than the controls. For a factor to confound a relationship it must also be associated with the exposure, both within the cases and within the controls. Stratifying the results by the case/control distinction (Table II*d*) illustrates a strong association between life events (the exposure) and the presence of children (the confounder) in this imaginary example.

TABLE II*a*

A hypothetical example of confounding: the relationship between schizophrenia and life events

	Cases	*Controls*
Life event	100	100
No life event	100	100
Total	200	200
	Odds ratio = 1.0	

TABLE II*b*

Stratifying the confounder: the presence of children at home

	Children		*No children*	
	Cases	*Controls*	*Cases*	*Controls*
Life event	45	95	55	5
No life event	5	50	95	50
Total	50	145	150	55
	Odds ratio = 4.7		Odds ratio = 5.8	

TABLE II*c*

Stratifying by the exposure: life events

	Life events		*No life events*	
	Cases	*Controls*	*Cases*	*Controls*
Children	45	95	5	50
No children	55	5	95	50
Total	100	100	100	100
	Odds ratio = 0.04		Odds ratio = 0.05	

TABLE II*d*

Stratifying by the disease: cases or controls

	Cases		*Controls*	
	LE+[1]	*LE−*[2]	*LE+*[1]	*LE−*[2]
Children	45	5	95	50
No children	55	95	5	50
Total	100	100	100	100
	Odds ratio = 15.5		Odds ratio = 19	

1. LE+, life events.
2. LE−, no life events.

Adjusting for confounders

Adjusting for confounders can occur in the design and/or the analysis but is only effective if they are measured accurately. Inaccurate measurement may result in a false sense of security, since residual confounding may occur despite the presence of a design or analysis that has apparently taken account of it. This can also occur if what is actually measured is an imperfect proxy for the true underlying confounder, for instance, if social class is used as a proxy for differences in lifestyle.

Often many (or even all) of the following four main methods of adjusting for confounding will be used in the same study.

(1) In the design – restriction

Subjects are only selected if they have the same value of the confounding variable. For instance, if sex is the confounder then only women may be selected for study (Brown & Harris, 1978). Jenkins (1985) investigated the sex difference in minor psychiatric morbidity by restricting her sample to one grade of the civil service thus reducing the confounding effects of occupational status and education. Controlling for confounding by restriction however makes it impossible to look for interactions (see below).

(2) In the design – matching

Each case can be matched with controls on the basis of any number of potential confounders, commonly age and sex. By matching, the investigator will have deliberately selected controls who, it is known, are different in regard to exposure from that found in a representative sample of the study population. This means that the matching must be retained in the analysis otherwise the estimate of relative risk may be biased towards unity. Matching can therefore *introduce* bias unless a matched analysis is performed.

Matching can either be on an individual basis or controls can be selected so that the proportion of those with the confounder in the controls conforms to that found in the cases. This is known as frequency matching. There are disadvantages and advantages to both methods (Rothman, 1986).

The main advantage of matching is that it increases statistical power. This is often useful if the disease is rare and cases are difficult to find. In addition, multiple controls for each case can be chosen, although there is usually little benefit in selecting more than four controls per case. However, individual matching can be unnecessarily time-consuming and expensive if cases are matched on several variables because many potential controls may have to be screened in order to find suitable ones.

Another disadvantage occurs if attempts to match on a potential confounder leads to a partial matching on the exposure under study. This is called overmatching because it *reduces* the power of the study.

(3) In the analysis – stratification

The principle of a stratified analysis is straightforward. The odds ratio is calculated within each level of the confounding variable and a summary statistic, a weighted average of some sort, is calculated. The details of analysis will not be described here, but the principle is illustrated in the hypothetical example given above (Table II*a* & II*b*). With stratification the relative risk of disease at each level of the confounding variable cannot be affected by the confounder.

(4) In the analysis – modelling techniques

It is often difficult to control for more than one or two confounders by stratification because the number of subjects in each stratum becomes very small or non-existent. For example, if the experimenter attempts to stratify by sex, age (in six age groups), and social class (six groups) there would be $2 \times 6 \times 6 = 72$ strata. Even with a large study there may be few or no cases or controls in certain strata and so those strata will not be of value in estimating relative risk.

Multivariate analysis is conceptually similar to stratification but makes a simplifying assumption which increases the apparent power of the study (Dunn, 1981). Case-control studies are usually analysed using logistic regression which assumes a linear relationship between the logarithm of the odds (logit) of being a case and the exposures. This model can therefore take account of a large number of variables simultaneously, including continuous measures.

However, there are problems with this method. It assumes a linear (or logistic) relationship (Warr, 1987, discusses non-linear relationships in mental health) and distances the investigator (and the reader) from the data being analysed. It is wise then to use linear modelling in conjunction with simpler stratified analysis.

Interactions or effect modification

Some factors may influence the size of an effect in a different way from that produced by a confounder. Interaction or effect modification occurs when the value of an exposure alters the size of the association between the disease and another risk factor. This is illustrated in Table III with results from a community survey (Bebbington *et al*, 1984). The study was examining the relationship between life events and

TABLE III
An example of an interaction from Bebbington et al *(1984)*

	Working class		*Middle class*	
	Cases	*Controls*	*Cases*	*Controls*
Life events	15	18	4	15
No life events	8	40	8	33
	Odds ratio = 4.17		Odds ratio = 1.10	

the onset of depression. Table III shows the results when they have been stratified by social class. It appears that the association between depression and life events is only apparent in the working-class group. Social class is therefore described as an effect modifier, or alternatively that there is an interaction between life events and low social class in increasing the likelihood of developing depression.

It is useful to compare Table III with the hypothetical example given in Table II*b*. In the latter, when the results are stratified by the presence of children, the odds ratios between life events and the disease are quite similar in the strata – there is no interaction.

Selection bias

Epidemiology is the population-based study of disease and the case-control study is no exception to this rule. The rationale of the case-control design is that the control group should give an estimate of the prevalence of the *exposure in the population from which the cases have been drawn*. Simple algebra illustrates that only under these circumstances will the odds ratio derived from a case-control study conform with the 'true' odds ratio in the study population (Schlesselman, 1982, p. 36). For this reason choosing the control group becomes one of the major problems in this study design. Selection bias occurs when the controls give a biased estimate of the prevalence of exposure in the study population.

Recently, it has been suggested that selection bias may be important in explaining the different results found when examining the association between enlarged cerebral ventricles and schizophrenia (Smith *et al*, 1988). These authors suggested that the variation between studies could be explained by differences in the control groups rather than from differences among the samples of schizophrenic patients.

The issue of selection bias makes the response rate of the study an issue. If a large proportion of the original sample of either cases or controls cannot be contacted then it is possible that the characteristics of those included in the final study will not be representative of the study population. Selection bias will only result if the likelihood of contacting subjects depends on the exposure.

Choosing cases

One consideration is that possible cases should be referrable to a study population. For instance, in a specialised hospital, restricting cases to those of the catchment area may make it easier to choose an appropriate control group.

Choosing a control group

A useful guide for choosing controls is that a control should become part of the sampling frame for the cases if he/she were to develop the disease. For instance, if the cases are all first-contact schizophrenics from a defined catchment area, it would be reasonable to choose a population-based sample from the catchment area as controls. This assumes that everyone with schizophrenia in the catchment area eventually contacts the psychiatric services.

The situation with affective disorder is quite different. There is considerable literature illustrating that the majority of those with affective disorder never contact the psychiatric services (Shepherd *et al*, 1966; Goldberg & Huxley, 1980). Therefore, general population controls for hospital cases of depression could lead to an important selection bias (see example below).

The sampling frame for controls will be chosen partly for practical reasons but will mainly depend on the cases under study and the hypothesis being explored. Several possible sources of controls are considered here.

(1) Clinic or hospital controls

The control group often consists of patients attending the same facility who have been diagnosed as suffering from a psychiatric disorder different from that under study. These subjects provide an unbiased estimate of the exposure in the study population, so long as the exposure is not associated with the control illness and the probability that controls are selected is not influenced by exposure.

It is desirable to avoid using control subjects with one condition. Thus, if there is an association between one control illness and the exposure, a large discrepancy in the relative risk estimate will be avoided.

(2) General population controls

General population controls are frequently used. If a serious psychiatric illness is under study and it is known that all, or almost all, cases in a particular area come to the attention of the local clinic, a general population control group may be the most appropriate.

In some investigations with an individually matched design, the population controls are the neighbours, friends or relatives of the cases (see, for example, Amaducci *et al*, 1986).

(3) Volunteer controls

Healthy volunteers are most commonly used in studies where a blood, imaging or other test is being investigated in psychiatric disorders. In this situation, it is difficult to assess the potential biases which may arise and, if volunteers are used, the investigators must make every attempt to describe who they were, why they volunteered, and what the response rate was.

In a recent study exploring positron emission tomography (PET) scan abnormalities in obsessive–compulsive disorder, no details were given on who the controls were and whether they differed markedly from the study population (Baxter *et al*, 1987). The general point made above, that controls should be included in the sampling frame for cases if they become diseased, tends to outlaw the use of volunteer controls in most psychiatric centres.

(4) Case-control studies and surveys

The widely acknowledged problem of selection bias in studying affective disorder has led to the adoption of community surveys to investigate the disorder. This ensures that both the cases and controls come from the same population and reduces the selection bias. Fortunately, many psychiatric conditions are common enough to adopt this approach, but in studying rarer disorders such as schizophrenia or severe depressions, surveys share the same disadvantage as cohort studies in requiring large numbers of subjects to detect small numbers of cases.

In studying common minor psychiatric disorders, one approach may be to screen a population and study a smaller sample of likely cases and controls in more depth. This has been called a two-stage screening procedure (Williams *et al*, 1980; Bebbington *et al*, 1981).

Examples of selection bias

Brown & Harris (1978) interviewed a sample of in-patients and out-patients in addition to a general population sample. The results (Table IV) illustrate that the association between having three or more children under 14 years at home and the onset of depression was only apparent when the general population cases were compared with the community controls (odds ratio = 3.77) and not when patient cases were compared with them. This is an example of selection into treatment. The presence of young children may increase the risk of depression, and also reduce the risk of receiving psychiatric care.

TABLE IV

An example of selection into treatment (from Brown & Harris (1978)): presence of more than 3 children (<14 years) in patient and general population cases of depression as well as in general population controls

		Patient cases	*General population cases*	*General population controls*
>3 children	yes	9	9	30
(<14 years)	no	105	28	352

Odds ratios comparing cases with general population controls: for patient cases = 1.01 (95% CI: 0.47, 2.19); for general population cases = 3.77 (95% CI: 1.63, 8.72).

The study by McGuffin *et al* (1988) may also be subject to selection bias. They noted an increase in the incidence of life events in the relatives of depressed hospital patients compared with the results of a population survey. Their results though suggest that the relatives were not very representative, because there was a marked preponderance of women (145:99). In addition, many relatives were not living in the Camberwell area when the study was conducted and so their comparison with Camberwell community data is less than ideal. Furthermore, they began by choosing relatives of depressed hospital patients who are unrepresentative of depressed people in the community. A case-control study could try to minimise the possible selection bias by choosing an appropriate control group.

Information bias

Psychiatric researchers have long been concerned with measurement because of the serious distortions that can result from inaccurate or biased assessment.

A distinction is made between random and systematic error. Random error will tend to bias the relative risk towards unity. A more serious problem arises from a systematic error: when there is a difference between the cases and controls in the gathering of data on disease or exposure. Such systematic error could lead to either an *increase* or *decrease* in the relative risk.

It is convenient to divide information bias into that derived from the subject and that from the observer although more exhaustive classifications exist (see Sackett, 1979).

Subject bias

Most case-control studies ask about exposure retrospectively and this leads to a serious source of bias, often termed 'recall bias': that subjects' recall of exposure may be influenced by their disease status.

One of the best known examples of recall bias is Stott's (1958) finding that mothers of children with Down's syndrome more frequently reported an emotional stressor during pregnancy than women who delivered a healthy baby. This is now thought to be due to the mothers' attempt to explain bearing a handicapped child – an 'effort after meaning'.

Recall bias is of importance in life-events research. A depressed subject may remember aversive life events more readily than someone without depression (Lloyd & Lishman, 1976). This would increase the apparent association between life events and depression.

There are several strategies for minimising recall bias and these should be considered at the design stage.

(a) Using a structured questionnaire and standardising the criteria for exposure is a prerequisite for reducing information bias, including recall bias. Relying upon recognition rather than recall by supplying the subject with a comprehensive series of prompts should also minimise this bias. This method is now routinely adopted in life-events research.
(b) Since recall bias is less likely to occur if the illness is of recent onset, the study of incident rather than prevalent cases is favoured.
(c) Control subjects who are themselves suffering from an illness may be equally motivated to search their memories with regard to exposure. A more appropriate comparison in the Down's syndrome study above would have been with the mothers of babies with a different handicap of known cause rather than the mothers of healthy babies.
(d) The validity of self-reported exposure data can be tested, for example by interviewing a close relative or consulting medical records.
(e) Raphael (1987) suggests that if other intuitively plausible exposures, which are known not to be risk factors, are not differentially recalled by the study groups, this lends weight to a finding of association with the hypothesised exposure. In addition, subjects could be asked about their own causal hypothesis, to see if this corresponds with that of the experimenter.

Social network (Henderson *et al*, 1981), personality measures (Katz & McGuffin, 1987), early aversive experiences (Wolkind & Coleman, 1983) and marital discord are probably influenced by current mental state. Therefore, case-control methodology and cross-sectional surveys may lead to misleading results. One potential approach is to design a case-control study within a cohort. For instance, Brown *et al* (1987) followed a group of women who were at increased risk of becoming depressed and collected data liable to be influenced by mental state at the beginning of the follow-up period. They then compared those who became depressed with those who did not using a case-control design. A similar design is often adopted (e.g. Kumar & Robson, 1984) in attempting to study risk factors for post-natal depression.

Observer bias

When an interviewer knows the subject is a case there may be biased assessment of the exposure, for instance by more careful probing. Similarly, knowledge of exposure, for instance a family member with mental illness, may influence diagnosis in doubtful cases. For this reason, epidemiologists have long argued against an over-reliance on unstructured clinical judgements in defining disease and exposure. The concept of 'blind' assessment in clinical trials (Hill, 1965) arose from the observation that knowledge of the treatment condition can seriously bias the results of a clinical assessment. Similar standards must also pertain to work studying associations in observational studies and so 'blind' assessments should be made whenever possible. In practice, this is often difficult because the author of the research proposal also acts as interviewer or because it is hard to sustain the interest and morale of the research team if they are unaware of the study purpose.

Epidemiologists who investigate the aetiology of physical diseases prefer to use non-medically trained interviewers as they are more likely to follow the questionnaire exactly and not to use their own judgement in deciding on exposure status. Another possibility is to tape-record interviews which can then be co-rated, preferably by someone who can be kept unaware of whether the subject is a case or a control. In research using imaging or laboratory techniques it is relatively easy for assessments to be performed by a rater who is unaware of whether the subject is a case or a control.

Observer bias is eliminated by self-report questionnaires and computerised assessments (Lewis *et al*, 1988). On the other hand, there is concern that subjects may not fully understand self-report questionnaires, and that those who read poorly or are illiterate may be unable to respond. Other sources of bias, for instance when the subject's responses are influenced by disease status (see subject bias), will remain when using self-report measures. Nevertheless, the reliance on clinical judgement in defining cases of psychiatric disorder in most psychiatric interviews is an unresolved problem as it may increase observer bias.

Measuring bias

If it is possible to measure a bias then it can be treated as a confounder. Unquantifiable biases are a major problem because it becomes difficult to interpret the results of a study. A bias may have a trivial effect or alternatively lead to a major distortion. The only solution to this difficulty is to attempt to measure bias, although "efforts to compensate for it [bias] in the analysis must be viewed with suspicion" (Rose & Barker, 1978).

An example of possible information bias

Goldberg & Morrison (1963) compared the social class of the parents of schizophrenic patients with that of the general population. As discussed above, the general population is probably an appropriate sample to compare with schizophrenic first admissions. However, they ascertained parental social class from the birth certificates of the schizophrenics and compared this with census data on the general population. Social class was therefore collected in different ways in cases and 'controls'. This can result in a well documented bias as social class tends to be 'higher' when recorded on birth certificates than when recorded by census (Leete & Fox, 1977). This suggests that a traditional case-control design would be more appropriate and would enable the social class of both cases and controls to be ascertained in the same way.

Conclusion

It should be clear that almost all the problems can (or should) be foreseen in the design of the study and the main intellectual challenge of conducting a study comes before the data are collected; in choosing the right control group, deciding on potential confounders and their appropriate measurement and assessing the importance of biases. It is therefore invaluable to consult colleagues at this stage and approach an epidemiologist and statistician, including those who might work outside the field of psychiatry.

Many authors feel reluctant to discuss the weaknesses of their own experimental design. This understandable but unfortunate failing hinders scientific progress. Only by an open discussion of weaknesses as well as strengths can new strategies be developed to overcome methodological problems.

Case-control studies provide both a challenge and an opportunity to the researcher. This has been well put by Breslow & Day (1980): "as a form of research the case-control study continues to offer a paradox: compared with other designs it is a rapid and efficient way to evaluate a hypothesis. On the other hand, despite its practicality, the case-control study is not simplistic and it cannot be done well without considerable planning. Indeed, the case-control study is perhaps the most challenging to design and conduct in such a way that bias is avoided. Our limited understanding of this difficult study design and its many subtleties should serve as a warning – these studies must be designed and analysed carefully with a thorough appreciation of their difficulties."

Appendix

Simple calculations in unmatched case-control studies

The odds ratio, R, and its confidence intervals will be calculated. Most authors will also carry out a χ^2 test of statistical significance. Table V shows the results of an unmatched case-control study in algebraic form. The odds ratio, which conforms to the relative risk as long as the condition is relatively rare, is calculated as follows:

Odds of being a case in exposed group = a/b
Odds of being a case in unexposed group = c/d
Odds ratio (exposed/unexposed) = (a/b)/(c/d) = ad/bc

There are a number of ways of calculating the confidence intervals; Woolf's method is given here.

The logarithm of the odds ratio (ln R) is approximately normally distributed. 95% confidence intervals for the odds ratio can then be calculated as follows:

$$\ln R \pm 1.96 \times \sqrt{(1/a + 1/b + 1/c + 1/d)}$$

Antilogs of the resulting limits give upper and lower 95% confidence limits for the odds ratio. When any cell in the table equals zero, 0.5 should be added to each cell before the variance of the logarithm of the odds ratio is calculated.

Matched case-control studies

Different methods of analysis are needed for matched case-control studies and conditional logistic regression is needed for multivariate analysis.

TABLE V
General 2×2 table showing the frequency of exposure in an unmatched sample of n_1 cases and n_2 controls

	Cases	*Controls*	*Total*
Exposed	a	b	m_1
Not exposed	c	d	m_2
Total	n_1	n_2	n

Further accounts

A number of good textbooks exist that discuss case-control studies in more depth. Schlesselman (1982) deals exclusively with case-control studies while Hennekens & Buring (1987) and the more difficult Rothman (1986) also discuss other areas of epidemiology. Breslow & Day (1980) have written a detailed and authoritative book on the analysis of case-control studies.

Acknowledgements

We would like to thank Professor Geoffrey Rose, Professor Michael Shepherd and the anonymous referees for comments. We also thank Professor Rose and his colleagues at the London School of Hygiene and Tropical Medicine for their teaching. The General Practice Research Unit is funded by the Department of Health and was under the direction of Professor Michael Shepherd. GL is funded by the Health Promotion Research Trust. AJP is a Wellcome Research Fellow in Clinical Epidemiology.

References

Amaducci, L. A., Fratiglioni, L., Rocca, W., *et al* (1986) Risk factors for clinically diagnosed Alzheimer's disease: a case-control study of an Italian population. *Neurology*, **36**, 922–931.

Andreassen, S., Allebeck, P., Engstrom, A., *et al* (1987) Cannabis and schizophrenia: a longitudinal study of Swedish conscripts. *Lancet*, *ii*, 1483–1485.

Baxter, L. R., Phelps, M. E., Mazziota, J. C., *et al* (1987) Local cerebral glucose metabolic rates in obsessive-compulsive disorder. *Archives of General Psychiatry*, **44**, 211–218.

Bebbington, P., Hurry, J., Tennant, C., *et al* (1981) Epidemiology of mental disorders in Camberwell. *Psychological Medicine*, **11**, 561–579.

——, Sturt, E., Tennant, C., *et al* (1984) Misfortune and resilience: a community study of women. *Psychological Medicine*, **14**, 347–363.

Breslow, N. E. & Day, N. E. (1980) *Statistical Methods in Cancer Research: Vol. 1, The Analysis of Case-Control Studies*. Oxford: Oxford University Press.

Brown, G. W. & Harris, T. (1978) *Social Origins of Depression*. London: Tavistock.

——, Bifulco, A. & Harris, T. O. (1987) Life events, vulnerability and the onset of depression: some refinements. *British Journal of Psychiatry*, **150**, 30–42.

Carroll, B. J., Feinberg, M., Greden, J. F., *et al* (1981) A specific laboratory test for the diagnosis of melancholia. *Archives of General Psychiatry*, **38**, 15–22.

Coryell, W. & Tsuang, M. T. (1982) DSM–III schizophreniform disorder. *Archives of General Psychiatry*, **39**, 66–69.

Craig, T. K. & Brown, G. W. (1984) Goal frustrating aspects of life events, stress and aetiology of gastro-intestinal disorder. *Journal of Psychosomatic Research*, **28**, 411–421.

Doll, R. & Hill, A. B. (1952) A study of the aetiology of carcinoma of the lung. *British Medical Journal*, *ii*, 1271–1286.

Dunn, G. (1981) The role of linear models in psychiatric epidemiology. *Psychological Medicine*, **11**, 179–184.

Eaton, W. W. (1985) Epidemiology of schizophrenia. *Epidemiologic Reviews*, **7**, 105–126.

Gardner, M. J. & Altman, D. G. (1986) Confidence intervals rather than P values: estimation rather than hypothesis testing. *British Medical Journal*, **292**, 746–750.

Goldberg, D. & Huxley, P. (1980) *Mental Illness in the Community*. London: Tavistock.

Goldberg, E. M. & Morrison, S. L. (1963) Schizophrenia and social class. *British Journal of Psychiatry*, **109**, 785–802.

Henderson, A. S., Byrne, D. G. & Duncan-Jones, P. (1981) *Neurosis and the Social Environment*. Sydney: Academic Press.

Hennekens, C. H. & Buring, J. E. (1987) *Epidemiology in Medicine*. Boston: Little Brown.

Hill, A. B. (1965) The environment and disease: association or causation? *Journal of the Royal Society of Medicine*, **58**, 295–300.

Jenkins, R. (1985) Sex differences in minor psychiatric disorder. *Psychological Medicine*, monograph no. 7.

Katz, R. & McGuffin, P. (1987) Neuroticism in familial depression. *Psychological Medicine*, **17**, 155–161.

Kumar, R. & Robson, K. M. (1984) A prospective study of emotional disorders in childbearing women. *British Journal of Psychiatry*, **144**, 35–47.

Leete, R. & Fox, J. (1977) Registrar General's social classes: origins and uses. *Population Trends*, **8**, 1–7.

Lewis, G. H., Pelosi, A. H., Glover, E., *et al* (1988) The development of a computerised assessment for minor psychiatric disorder. *Psychological Medicine*, **18**, 737–745.

Lewis, S.W. & Murray, R. M. (1987) Obstetric complications, neurodevelopmental deviance and risk of schizophrenia. *Journal of Psychiatric Research*, **21**, 413–421.

Lloyd, G. & Lishman, A. (1976) The effect of depression on speed of recall of pleasant and unpleasant experiences. *Psychological Medicine*, **5**, 173–180.

Mantel, N. & Haenzel, W. (1959) Statistical aspects of the analysis of data from retrospective studies of disease. *Journal of the National Cancer Institute*, **22**, 719–748.

McGuffin, P., Katz, R., Aldrich, J., *et al* (1988) The Camberwell collaborative depression study. II: Investigation of family members. *British Journal of Psychiatry*, **152**, 766–774.

Mukherjee, S., Rosen, A. M., Caracci, G., *et al* (1986) Persistent tardive dyskinesia in bipolar patients. *Archives of General Psychiatry*, **43**, 342–346.

Mullen, P. E., Linsell, C. R. & Parker, D. (1986) Influence of sleep disruption and calorie restriction on biological markers for depression. *Lancet*, *ii*, 1051–1054.

Raphael, K. (1987) Recall bias: a proposal for assessment for control. *International Journal of Epidemiology*, **16**, 167–170.

Rose, G. & Barker, D. J. P. (1978) Observer variation. *British Medical Journal*, *ii*, 1006–1007.

Rothman, K. (1986) *Modern Epidemiology*. Boston: Little Brown.

Sackett, D. L. (1979) Bias in analytic research. *Journal of Chronic Diseases*, **32**, 51–63.

Schlesselman, J. J. (1982) *Case Control Studies*. New York: Oxford University Press.

Shepherd, M., Cooper, B., Brown, A. C., *et al* (1966) *Psychiatric Illness in General Practice*. Oxford: Oxford University Press

Smith, G. N., Iacono, W. G., Moreau, M., *et al* (1988) Choice of comparison group and findings of computerised tomography in schizophrenia. *British Journal of Psychiatry*, **153**, 667–674.

Smith, P. G., Rodrigues, L. C. & Fine, P. E. M. (1984) Assessment of the protective efficacy of vaccines against common diseases using case-control and cohort studies. *American Journal of Epidemiology*, **13**, 87–93.

Stott, D. H. (1958) Some psychosomatic aspects of causality in reproduction. *Journal of Psychosomatic Research*, **3**, 42–55.

Warr, P. (1987) *Work, Unemployment and Mental Health* Oxford: Oxford University Press.

Williams, P., Tarnopolsky, A. & Hand, D. (1980) Case definition and case identification in psychiatric epidemiology: review and assessment. *Psychological Medicine*, **10**, 101–114.

Wolkind, S. & Coleman, E. Z. (1983) Adult psychiatric disorder and childhood experiences: the validity of retrospective data. *British Journal of Psychiatry*, **143**, 188–191.

*Glyn Lewis, MSc, MRCPsych, *Lecturer, General Practice Research Unit, Institute of Psychiatry*; Anthony J. Pelosi, MSc, MRCP, MRCPsych, *Research Fellow in Clinical Epidemiology, Institute of Psychiatry*

*Correspondence: *Institute of Psychiatry, DeCrespigny Park, London SE5 8AF*

British Journal of Psychiatry (1990), 157, 208–215

^{3}H-Imipramine Binding to Previously Frozen Platelet Membranes from Depressed Patients, Before and After Treatment

DAVID HEALY, ANDREAS E. THEODOROU, ANDREW M. WHITEHOUSE, KEVIN M. LAWRENCE, WILLIAM WHITE, HELEN WILTON-COX, SALLY M. KERRY, ROGER W. HORTON and EUGENE S. PAYKEL

^{3}H-imipramine binding in 39 drug-free patients with major depression and 44 healthy controls did not differ significantly between the two groups, in male or female subjects or in subgroups of depressed patients divided by endogenicity or dexamethasone suppression test result. ^{3}H-imipramine binding in depressed patients drug-free for less than three weeks did not differ from those drug-free for longer intervals or from controls. A significant seasonal variation of ^{3}H-imipramine *B*max was found, with lower values in summer and autumn. Treatment of depressed patients with imipramine or lofepramine for six weeks increased K_D and *B*max. Methodological modification (in preparation and storage of platelets) does not explain the major differences in results between this study (using frozen platelets), a previous one (using freshly prepared platelets) and others in general, although it might contribute to the range of values reported.

Specific high-affinity binding sites for ^{3}H-imipramine (^{3}H-IMIP) are associated with the serotonin uptake mechanism in neurones and platelets (Langer *et al*, 1982). Initial studies reported markedly lower numbers of platelet ^{3}H-IMIP binding sites (reduced *B*max) in depressed patients compared with controls (Briley *et al*, 1980; Raisman *et al*, 1981, 1982). Some subsequent studies have replicated these findings (Poirier *et al*, 1986; Innis *et al*, 1987; Maj *et al*, 1988), others have reported less marked but statistically significant reductions (Lewis & McChesney, 1985; Wägner *et al*, 1985) and an increasing number have found no significant differences between depressed patients and controls (Gentsch *et al*, 1985; Hrdina *et al*, 1985; Carstens *et al*, 1986; Braddock *et al*, 1986; Roy *et al*, 1987; Plenge *et al*, 1988). A recent World Health Organization (WHO) multicentre pilot study also reported no such differences (Bech *et al*, 1988).

The discrepancies between studies cast doubts on the validity of reduced platelet ^{3}H-IMIP as a useful biological marker of depression and therefore it is important to attempt to identify the factors which contribute to the inconsistencies. Subject selection may be important. The depressed patients studied have varied widely in their severity and symptoms. Some studies suggest that reduced *B*max of platelet ^{3}H-IMIP binding is associated with certain subgroups of depressed patients, for example those with a positive family history of depression (Lewis & McChesney, 1985; Schneider *et al*, 1986) or with specific clinical symptoms, such as motor retardation (Carstens *et al*, 1986) or psychotic features (Arora *et al*, 1985). Furthermore, since some antidepressants produce changes in platelet ^{3}H-IMIP binding that persist for several weeks following drug withdrawal (Braddock *et al*, 1984; Poirier *et al*, 1987), the length of time patients were free of these drugs before blood sampling may be a significant factor. Seasonal variations in platelet ^{3}H-IMIP binding, although inconsistently reported, may also influence results.

Furthermore, methodological differences may contribute to both the discrepancies between studies in depression and the large variations in equilibrium dissociation constants (K_D) and number of binding sites (*B*max) values reported for control subjects between laboratories (Mellerup & Plenge, 1988). For example, platelets have been isolated by different methods, and whole platelets and membranes, from freshly prepared and previously frozen platelets, have been studied.

We have recently reported a study of ^{3}H-IMIP binding in a large group of depressed patients (Theodorou *et al*, 1989). Platelets were isolated and platelet membranes prepared and assayed on the same day. We found that overall ^{3}H-IMIP *B*max did not differ significantly between depressed patients and controls. There was a small (14%) but significant reduction in female depressed patients, but not in males. Lower ^{3}H-IMIP *B*max was not associated with endogenicity, dexamethasone suppression test result, family history of depression, depressive psychosis, suicide ideation or past history of suicide attempts. ^{3}H-IMIP *B*max was however lower in those depressed patients with obsessional features.

We now report a further study of platelet ^{3}H-IMIP binding in which we have addressed the effects of platelet isolation and storage in a new cohort of depressed patients and controls. Two aspects of our previous methodology were altered. Firstly, platelet isolation was modified to increase platelet yield and to increase recovery of heavy platelets. Secondly, platelets were stored frozen before assay, to allow a more direct comparison with the majority of published studies.

Method

Depressed patients were recruited from in-patients ($n = 28$) at the Professorial Unit, Fulbourn Hospital, Cambridge or out-patients ($n = 11$) attending the Affective Disorders Clinic, Addenbrooke's Hospital, Cambridge between June 1986 and December 1987. Patients were entered into the study, subject to their informed written consent, if they fulfilled the following criteria: (1) Research Diagnostic Criteria (RDC; Spitzer & Endicott, 1978) for major depressive disorder, probable or definite, or for bipolar depression; (2) a minimum score of 17 on the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967); (3) no major physical illness; (4) at least seven days free of antidepressant drugs and at least six months free from neuroleptics. Particular efforts were made to recruit patients who were antidepressant-free for six weeks or more. A number of patients continued to receive short-acting benzodiazepines for night-time sedation.

Controls were recruited over the same time period from among the staff of the psychiatric services and their acquaintances. They had no physical or psychiatric illnesses and were free of psychoactive drugs.

Psychiatric assessments

These included: (1) detailed demographic, social, family and past psychiatric history questionnaires; (2) ratings of depressive severity using the HRSD and the Clinical Interview for Depression (CID; Paykel, 1985); (3) a rating of psychopathology using the Present State Examination (PSE; Wing *et al*, 1974); (4) a rating of endogenicity using the Newcastle Depression Scale (NDS; Carney *et al*, 1965) (endogenicity defined as a NDS score >5); (5) depressive diagnoses using the RDC and the International Classification of Diseases (ICD-9; World Health Organisation, 1975). These assessments were carried out at baseline, and HRSD and CID were repeated after six weeks of treatment.

Treatment

Patients were treated for up to six weeks with imipramine (150–225 mg/day), lofepramine (140–210 mg/day) or electroconvulsive therapy (ECT). It was not possible to obtain post-treatment results on all patients as some required additional treatment to the above, some left care and, in a small number of cases, assays were unsuccessful.

^{3}H-IMIP binding to platelet membranes

Baseline blood samples of 30 ml were taken by antecubital venepuncture between 09:00 and 10:00 hours and immediately mixed with 3 ml of acid-citrate-dextrose to prevent coagulation. Platelets were isolated by a modification of Corash's (1980) method. Following centrifugation at 100 *g* for 10 min, platelet-rich plasma was removed by aspiration and the original blood volume restored with normal saline. Centrifugation was repeated as above and the supernatant collected. The reconstitution with normal saline and centrifugation were repeated one further time. The original platelet-rich plasma and the saline supernatants were combined and centrifuged at 1500 *g* for 15 min. The supernatant was discarded, the platelet pellet rapidly frozen by immersion in a dry-ice alcohol mixture and stored at −80°C until assayed.

On the day of assay, 5 ml of washing buffer (50 mM TRIS-HCl (*tris*(hydroxymethyl)aminomethane-hydrochloric acid), 110 mM sodium chloride (NaCl), 20 mM ethylenediaminetetra-acetic acid (EDTA), pH 6.4) was added to the platelet pellets and the platelet suspension stood on ice for 5 min before being homogenised (six strokes, teflon in glass, motor driven at 800 r.p.m.) and centrifuged at 18 500 *g* for 10 min. The supernatant was discarded and the pellet homogenised and centrifuged as above. The supernatant was discarded and the pellet homogenised (12 strokes at 1200 r.p.m.) in hypotonic lysing buffer (5 mM *tris*- HCl, 5 mM EDTA, pH 7.4), allowed to stand on ice for 20 min, and centrifuged at 42 000 *g* for 10 min. The pellet was suspended in incubation buffer (50 mM *tris*-HCl, 120 mM NaCl, 5 mM potassium chloride (KCl), pH 7.5) and centrifuged at 42 000 *g* for 10 min. The resulting pellet was resuspended in incubation buffer (to give 50–70 μg protein/assay tube) and incubated with nine concentrations (0.2–6 nM) of ^{3}H-IMIP (specific radioactivity 20 Ci/mmol) for 75 min at 0°C in a volume of 0.5 ml. Specific binding was defined as radioactivity displaced by 10^{-4} M desmethylimipramine. Membrane-bound radioactivity was recovered by filtration under reduced pressure through Whatman glass-fibre (GF/C) filters, presoaked in 0.5% polyethylenimine to reduce non-specific filter binding. Filters were washed (four times, 4 ml) with ice-cold incubation buffer, placed in scintillation vials containing 6 ml of Packard ES 299 scintillation cocktail and counted at an efficiency of 38–45%. Samples from depressed patients and controls were assayed concurrently. Aliquots of membrane were stored at −20°C before protein determination by the method of Lowry *et al* (1951) using bovine serum albumen as a standard.

Both *B*max and K_D were determined by computerised non-linear fitting (using a commercial package, Statistical Analytical Systems, 1982) to saturation plots.

Dexamethasone suppression test (DST)

Dexamethasone (1 mg) was administered orally to depressed patients at 20:00 hours on the same day as the baseline blood sample for platelet ^{3}H-IMIP binding was taken. Blood (10 ml) was taken by antecubital venepuncture at 15:30 hours on the following day, placed in a heparinised tube and immediately centrifuged at 4000 *g* for 4 min.

TABLE I

Clinical features of depressed subjects, based on Research Diagnostic Criteria (RDC), Newcastle Depression Scale (NDS), dexamethasone suppression test (DST) and Hamilton Rating Scale for Depression (HRSD)

	Total (n=39)	*Female (n=27)*	*Male (n=12)*
RDC definite endogenous: %	75	74	75
NDS endogenous: %	54	52	58
DST non-suppressors[1]: %	42	50	17
Baseline HRSD total: mean (s.e.m.)	26 (1.0)	27 (1.2)	25 (2.0)

1. DST was carried out in 20 females and 6 males.

Plasma was frozen at −20°C before cortisol determination by radio-immunoassay using a commercial kit (*Amerlex*, Amersham International, UK). Non-suppression was defined as a post-dexamethasone cortisol of ≥5 ug/dl. The DST was not performed on out-patients.

Statistical analysis

*B*max values approximated to a normal distribution. K_D values approximated to a normal distribution following log transformation, hence statistical tests on K_D values were carried out using log transformed data. Differences between pairs of means were compared by Student's *t*-test (two-tailed, unpaired except two-tailed, paired was used for the effects of treatment). For multiple comparisons, the overall differences between means were first compared using *F*-tests on the one-way analysis of variance, followed by *t*-tests (three groups) or Gabriel's test (more than three groups).

Results

The clinical features of the depressed patients are shown in Table I.

Comparison between depressed patients and controls

*B*max and K_D of ^{3}H-IMIP binding did not differ significantly between controls and depressed patients in the total group or when divided by sex (Table II) or in the female subjects pre- or post-menopausally (data not shown).

Comparison between depressive subgroups and controls

*B*max and K_D of ^{3}H-IMIP binding did not differ significantly between NDS- or RDC-defined endogenous and non-endogenous subgroups (Table II) and DST suppressors or non-suppressors (Table III) or between these subgroups and controls in the total sample and in female subjects.

Specific clinical features

Depressed subjects were divided in terms of past history of suicide attempts, serious suicide attempts and current suicidal intent, obsessional features (HRSD and CID), diurnal mood variation (HRSD and CID), weight loss (HRSD) reactivity of mood (CID) and depressive delusions (CID).

Depressed patients with high scores on suicide ideation and those who had a past history of attempted suicide or

TABLE II

^{3}H-IMIP binding parameters (mean values (s.e.m.)) in controls and diagnostically defined (Research Diagnostic Criteria (RDC), Newcastle Depression Scale (NDS)) depressives – total sample and divided by sex

	Age	K_D: n*M*	*B*max: *fmol/mg protein*
All subjects			
Controls (n=44)	40.5 (1.7)	0.56 (0.04)	1076 (45)
Depressed (n=39)	47.1 (2.3)	0.55 (0.03)	1087 (45)
RDC endogenous (29)	45.6 (2.9)	0.57 (0.04)	1071 (54)
RDC non-endogenous (10)	51.4 (3.5)	0.49 (0.03)	1133 (80)
NDS endogenous (21)	48.7 (3.6)	0.54 (0.04)	1044 (57)
NDS non-endogenous (18)	45.2 (2.8)	0.55 (0.04)	1137 (70)
Female subjects			
Controls (n=26)	40.1 (2.2)	0.57 (0.06)	1050 (50)
Depressed (n=27)	43.9 (2.6)	0.55 (0.04)	1084 (44)
RDC endogenous (20)	42.0 (3.3)	0.59 (0.05)	1044 (51)
RDC non-endogenous (7)	49.1 (3.8)	0.46 (0.04)	1199 (74)
NDS endogenous (14)	44.7 (4.2)	0.58 (0.06)	1062 (66)
NDS non-endogenous (13)	42.9 (3.2)	0.53 (0.05)	1109 (59)
Male subjects			
Controls (n=18)	41.0 (2.7)	0.55 (0.05)	1113 (85)
Depressed (n=12)	54.3 (4.1)	0.53 (0.04)	1092 (110)

TABLE III

^{3}H-IMIP binding parameters (mean values (s.e.m.)) and dexamethasone suppression test results

	Age	K_D: n*M*	B*max*: *fmol/mg protein*
All subjects			
Controls ($n = 44$)	40.5 (1.7)	0.56 (0.04)	1076 (45)
DST suppressors ($n = 15$)	47.0 (3.3)	0.50 (0.03)	1076 (60)
DST non-suppressors ($n = 11$)	49.5 (4.7)	0.55 (0.06)	1139 (64)
Female subjects			
Controls ($n = 26$)	40.1 (2.2)	0.56 (0.06)	1050 (50)
DST suppressors ($n = 10$)	43.8 (3.6)	0.52 (0.03)	1071 (63)
DST non-suppressors ($n = 10$)	48.1 (4.9)	0.54 (0.07)	1167 (64)

had made serious suicide attempts, tended to have higher ^{3}H-IMIP *B*max values than those without these features. *B*max was significantly higher in those patients who had made serious suicide attempts (1203 (s.e.m. 72) fmol/mg protein, $n = 16$) than those who had not (1006 (s.e.m. 52) fmol/mg protein, $n = 23$; $P < 0.035$), although neither group differed significantly from controls.

Depressed patients whose mood was significantly worse in the morning had lower ^{3}H-IMIP *B*max than those who had no diurnal mood variation or were worse in the afternoon (HRSD item, worse a.m., 946 (s.e.m. 47) fmol/mg protein, $n = 17$; others 1195 (s.e.m. 62), $n = 22$, $P < 0.01$; CID item, worse a.m., 1015 (s.e.m. 45) $n = 25$; others 1214 (s.e.m. 88), $n = 14$, $P < 0.05$). Neither of these groups differed significantly from controls.

*B*max values in depressed patients with obsessional features (HRSD obsessionality item ≥ 1, *B*max 1192 (s.e.m. 56) fmol/mg protein, $n = 13$; CID obsessionality score ≥ 4, *B*max 1145 (s.e.m. 60) fmol/mg protein, $n = 10$) did not differ significantly from patients without obsessional features (HRSD obsessionality item = 0, *B*max 1034 (s.e.m. 59) fmol/mg protein, $n = 26$; CID obsessionality score < 4, *B*max 1062 (s.e.m. 59) fmol/mg protein, $n = 28$). Again, neither of these groups differed significantly from controls.

*B*max and K_D did not differ significantly between depressed patients defined by weight loss, reactivity of mood or depressive delusions.

Drug-free interval before baseline testing

Depressed patients were divided according to the interval between previous antidepressant-drug treatment and blood sampling. K_D and *B*max of ^{3}H-IMIP binding did not differ significantly between those patients antidepressant-free for 7–21 days (K_D 0.48 (s.e.m. 0.03) nM, *B*max 1033 (s.e.m. 82) fmol/mg protein, $n = 11$) and those antidepressant-free for a minimum of three weeks (K_D 0.57 (s.e.m. 0.04) nM, *B*max 1108 (s.e.m. 54) fmol/mg protein, $n = 28$), or between either of these groups and control subjects.

TABLE IV

Effects of treatment on ^{3}H-IMIP binding parameters (mean values (s.e.m.)) in depressed patients

	K_D: *nM*	B*max*: *fmol/mg protein*
All subjects ($n = 18$)		
at baseline	0.48 (0.02)	1056 (59)
after 6 weeks' treatment	0.58 (0.04)	1227 (69)*
Subjects treated with imipramine or lofepramine ($n = 16$)		
at baseline	0.48 (0.02)	1075 (63)
after 6 weeks' treatment	0.59 (0.05)*	1256 (65)*
Subjects treated with imipramine ($n = 10$)		
at baseline	0.48 (0.03)	1161 (82)
after 6 weeks' treatment	0.57 (0.05)	1340 (80)
Subjects treated with lofepramine ($n = 6$)		
at baseline	0.47 (0.04)	932 (73)
after 6 weeks' treatment	0.62 (0.09)	1116 (92)

*$P < 0.05$ (*t*-test, two-tailed, paired) – differences between paired baseline and treatment values.

Mean values (s.e.m.) of K_D and *B*max in depressed patients at baseline and after six weeks of treatment.

Effects of treatment

In 18 of the depressed patients, ^{3}H-IMIP binding was performed at baseline and after six weeks of treatment. Treatment was associated with a significant increase in *B*max (Table IV). In depressed patients treated with imipramine ($n = 10$) or lofepramine ($n = 6$) increases in both *B*max and K_D were found, although for each drug the difference failed to reach statistical significance (Table IV).

Effects of season

Subjects were divided by season of testing. A significant seasonal variation in *B*max was seen in controls and depressed patients, with low values in summer and autumn (Table V). The seasonal variation in *B*max was of similar magnitude in male and female subjects (Table VI). No significant seasonal variation in K_D was seen (ANOVA; season, $F(3,75 = 0.82$, $P = 0.49)$.

TABLE V

Seasonal variation in mean Bmax values (s.e.m.) of platelet ^{3}H-IMIP in control and depressed subjects

Season	*All*	*Controls*	*Depressed*
Spring (Mar–May)	1309 (53) ($n=17$)	1267 (66) ($n=11$)	1385 (89) ($n=6$)
Summer (Jun–Aug)	1069 (66) ($n=11$)	987 (126) ($n=3$)	1099 (77) ($n=8$)
Autumn (Sep–Nov)	885 (35) ($n=38$)	887 (48) ($n=21$)	896 (53) ($n=17$)
Winter (Dec–Feb)	1299 (53) ($n=17$)	1337 (73) ($n=9$)	1255 (77) ($n=8$)

Analysis of variance revealed significant effects of season ($F(3,75)=21.99$, $P<0.001$) but not of illness after adjusting for season (depressed v. control, $F(1,75)=0.31$, $P=0.58$).

Discussion

In the present study we have examined ^{3}H-IMIP binding in a second large cohort of depressed patients and controls. The same subject selection criteria were applied as in our previous study (Theodorou *et al*, 1989) and although subjects were recruited from a different geographical area, the two populations were very similar both in their demographic characteristics and in the nature, severity and clinical features of their depression. Thus, differences between our present and previous findings (Theodorou *et al*, 1989) are likely to have arisen because of methodological factors, rather than differences in the subjects.

Two aspects of methodology were altered. Differences in platelet recovery may in part explain the variability of findings between studies, since larger and heavier platelets have more ^{3}H-IMIP binding sites than smaller, lighter platelets (Arora & Meltzer, 1984). Thus platelet-rich plasma was prepared by a method which maximises platelet yield and, in particular, recovers heavier platelets. The other difference from our previous study was that platelets were stored frozen before assay. We thought this might be important since our *B*max values in freshly prepared platelets were higher than those reported for frozen platelets.

*B*max values in controls were significantly lower than those we reported for fresh platelets (*B*max, 1668 fmol/mg protein; Theodorou *et al*, 1989). The substantial difference between fresh and frozen platelets seems unlikely to be attributable to platelet preparation, since the presumed greater yield of heavier platelets would be expected to increase, rather than decrease, ^{3}H-IMIP *B*max (Arora & Meltzer, 1984). The difference in *B*max may be related to freezing and/or storage of platelets before assay. *B*max values for controls in the present study were within the range reported for frozen platelets in some studies (Mellerup *et al*, 1982; Lewis & McChesney, 1985; Hrdina *et al*, 1985; Wägner *et al*, 1985; Plenge *et al*, 1988) but were higher than values in other studies (Briley *et al*, 1980; Paul *et al*, 1981; Raisman *et al*, 1981, 1982; Roy *et al*, 1987), suggesting that freezing/storage alone is not responsible for the differences. There are no studies in which ^{3}H-IMIP binding has been compared directly in fresh and frozen platelets from the same individuals.

K_D values have also differed widely between studies. Our K_D values in frozen platelets were lower than many others reported and lower than those we reported in fresh platelets. Mellerup & Plenge (1988) recently reviewed studies of platelet ^{3}H-IMIP binding in depression and concluded that under optimal assay conditions, K_D values should be 0.5 nM, close to the value of 0.56 nM that we found. Higher K_D values (lower binding affinity) are a reflection of non-optimal conditions. They commented that of the 19 studies that reported a significant reduction in *B*max in depression, 84% reported K_D values greater than 1 nM while of 11 studies that reported no difference in *B*max in depression only 45% had K_D values greater than 1 nM (Mellerup & Plenge, 1988).

TABLE VI

Seasonal variation in mean Bmax values (s.e.m.) of platelet ^{3}H-IMIP in male and female control and depressed subjects

	Males	*Females*
Spring (Mar–May)	1417 (80) ($n=7$)	1232 (67) ($n=10$)
Summer (Jun–Aug)	1070 (149) ($n=2$)	1069 (70) ($n=9$)
Autumn (Sep–Nov)	837 (54) ($n=15$)	917 (44) ($n=23$)
Winter (Dec–Feb)	1421 (86) ($n=6$)	1232 (64) ($n=11$)

Analysis of variance revealed no significant effect of gender after adjusting for season ($F(1,75)=0.59$, NS) or of the interaction term for gender x season ($F(3,75)=2.27$, NS).

As in our previous study we found no overall difference in K_D or Bmax of ^{3}H-IMIP binding between controls and depressed patients. The small but significantly lower number of ^{3}H-IMIP binding sites we previously reported in female depressed patients was not apparent (Theodorou *et al*, 1989).

The considerable overlap in Bmax values for patients and controls, apparent even in studies that demonstrate large differences in mean values, suggests that reduced Bmax of platelet ^{3}H-IMIP binding is restricted to a subgroup of depressed patients. However, in agreement with our previous findings (Theodorou *et al*, 1989), we found no difference in ^{3}H-IMIP binding between endogenous and non-endogenous depressed patients (defined by RDC or NDS) or between either of these groups and controls. Similar findings have been reported by others (Baron *et al*, 1986; Roy *et al*, 1987; Plenge *et al*, 1988).

It has also been suggested that reduced Bmax of ^{3}H-IMIP binding in depression is secondary to hypercortisolaemia (Roy *et al*, 1987), and we have previously reported DST non-suppressors to have lower ^{3}H-IMIP Bmax than controls (Theodorou *et al*, 1989). However, no such difference was found in the present study. Other studies have found no significant relationship between DST result or post-DST cortisol concentration and ^{3}H-IMIP binding (Carstens *et al*, 1986; Nemeroff *et al*, 1988). Thus, the evidence to date suggests that the major categorical variables of endogenicity and DST are not able to identify depressed patients in which ^{3}H-IMIP Bmax is reduced.

We have examined possible relationships between ^{3}H-IMIP binding and specific clinical symptoms within depressed patients. No relationship was found with weight loss, reactivity of mood and depressive delusions. Serious suicide attempts and diurnal mood variation were associated with increased and decreased ^{3}H-IMIP Bmax respectively, although in neither case were Bmax values in the presence or absence of the symptom different from control values. In our previous study no relationship was identified between these symptoms and ^{3}H-IMIP Bmax. However, in that study we did find that depressed patients with marked obsessional features had lower Bmax than both patients without such symptoms and controls. The present study did not replicate this finding; the tendency was in the opposite direction. Thus, although the present and our previous study (Theodorou *et al*, 1989) have each identified relationships between specific symptoms and ^{3}H-IMIP Bmax that satisfy criteria of statistical significance, the lack of replication between studies casts doubts on their importance.

Increases in platelet ^{3}H-IMIP binding have previously been reported in depressed patients following treatment with zimelidine, alaproclate and desipramine (Wagner *et al*, 1987; Arora & Meltzer, 1988), and in healthy volunteers following administration of amitriptyline and desipramine (Braddock *et al*, 1984; Cowen *et al*, 1986; Healy *et al*, 1990). In the present study, there were significant increases in K_D and Bmax of ^{3}H-IMIP binding in patients treated with imipramine or lofepramine for six weeks. This effect may not be common to all antidepressants. Short-term administration of nortriptyline in depressed patients (Wägner *et al*, 1987) or amineptine, maprotiline or imipramine in healthy controls (Suranyi-Cadotte *et al*, 1985; Poirier *et al*, 1987) was not associated with changes in ^{3}H-IMIP Bmax, and administration of chlorimipramine to healthy subjects for one week resulted in a marked decrease in ^{3}H-IMIP Bmax (Poirier *et al*, 1987). Clearly, effects of previous antidepressant treatment need to be considered when comparing baseline measures in depressed patients with controls.

Depressed patients included in this study varied widely in their recent drug history and in the antidepressant-free interval prior to baseline blood sampling. However, ^{3}H-IMIP Bmax in those depressed patients who were antidepressant-free for a minimum of three weeks before baseline testing, many of whom had never previously received antidepressants, did not differ from controls. Perhaps surprisingly in view of the above evidence, ^{3}H-IMIP Bmax in those depressed patients who were antidepressant-free for less than three weeks also did not differ significantly from those patients drug-free for longer intervals or from controls. The majority of these patients were drug-free for between two and three weeks (all were drug-free for a minimum of seven days) and this may have been sufficient time for residual effects of previous antidepressants to be no longer apparent.

Several studies have reported no seasonal variation in ^{3}H-IMIP binding (Carstens *et al*, 1986; Galzin *et al*, 1986), while others have reported significant, although varied, seasonal differences (Whitaker *et al*, 1984; Hrdina *et al*, 1985; Egrise *et al*, 1986; Kanof *et al*, 1987). Although the present study was not specifically designed to test for seasonal variations, we found significantly lower Bmax values in summer and autumn; an effect apparent in controls and depressed patients and in male and female subjects. These findings are similar to those reported by Whitaker *et al* (1984). It is possible that differences between controls and depressed patients could arise or be masked artefactually by the season of testing. However, this seems unlikely since controls and patients were recruited throughout the whole period of the study and, apart from some mismatch in the summer, were reasonably matched for season of testing.

Modifications in methodology indicate that although platelet preparation and storage affect ^{3}H-IMIP binding and thus contribute to the wide range of reported values, they do not explain the major differences between studies of depressed patients and controls. Our present and previous studies (Theodorou *et al*, 1989), in two separate patient and control populations totalling 86 patients and 90 controls, found no difference overall in K_D or Bmax of ^{3}H-IMIP binding in depression, and no reproducible relationship between reduced ^{3}H-IMIP Bmax and the major categorical variables of endogenicity and DST or with specific symptoms.

A biological marker for depression should be demonstrable and quantitatively reproducible between studies and between laboratories, allowing for diagnostic variability. Although some reports of ^{3}H-IMIP binding relate to rather small numbers of subjects and thus may lack the statistical power necessary to detect small differences between controls and depressed patients, there are now sufficient studies, with adequate numbers of subjects, to doubt the validity of marked reductions in platelet ^{3}H-IMIP binding in depression. Although the possible association of reduced Bmax with selected subgroups of depressed has some support, as yet such factors have not been clearly or consistently identified.

Acknowledgement

This study was supported by a Major Award from the Wellcome Trust.

References

Arora, R. C. & Meltzer, H. Y. (1984) Imipramine binding in subpopulations of normal human platelets. *Biological Psychiatry*, **19**, 257–262.

——, Wunnicke, V. & Meltzer, H. Y. (1985) Effect of protein concentration on kinetic constants (k_d and Bmax) of ^{3}H-imipramine binding in blood platelets. *Biological Psychiatry*, **20**, 116–119.

—— & Meltzer, H. Y. (1988) Effects of desipramine treatment on ^{3}H-imipramine binding in the blood platelets of depressed patients. *Biological Psychiatry*, **23**, 397–404.

Baron, M., Barkai, A., Gruen, R., *et al* (1986) Platelet [^{3}H] imipramine binding in affective disorders: trait versus state characteristics. *American Journal of Psychiatry*, **143**, 711–717.

Bech, P., Eplov, L., Gastpar, M., *et al* (1988) WHO pilot study on the validity of imipramine platelet receptor binding sites as a biological marker of endogenous depression. *Pharmacopsychiatry*, **21**, 147–150.

Braddock, L. E., Cowen, P. J., Elliott, J. M., *et al* (1984) Changes in the binding to platelets of [^{3}H]imipramine and [^{3}H]yohimbine in normal subjects taking amitriptyline. *Neuropharmacology*, **23**, 285–286.

——, Cowen, P. J., Elliott, J. M., *et al* (1986) Binding of yohimbine and imipramine to platelets in depressive illness. *Psychological Medicine*, **16**, 765–773.

Briley, M. S., Langer, S. Z., Raisman, R., *et al* (1980) Tritiated imipramine binding sites are decreased in platelets of untreated depressed patients. *Science*, **209**, 303–305.

Carney, M. W. P., Roth, M. & Garside, E. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **149**, 659–674.

Carstens, M. E., Engelbrecht, A. H., Russell, V. A., *et al* (1986) Imipramine binding sites on platelets of patients with major depressive disorder. *Psychiatry Research*, **18**, 333–342.

Corash, L. (1980) Platelet heterogeneity; relevance to the use of platelets to study psychiatric disorders. *Schizophrenia Bulletin*, **6**, 254–258.

Cowen, P. J., Geany, D. P., Schachter, M., *et al* (1986) Desipramine treatment in normal subjects. Effects on neuroendocrine responses to tryptophan and on platelet serotonin (5-HT)-related receptors. *Archives of General Psychiatry*, **43**, 61–67.

Egrise, D., Rubinstein, M., Schoutens, A., *et al* (1986) Seasonal variation of platelet serotonin uptake and ^{3}H-imipramine binding in normal and depressed subjects. *Biological Psychiatry*, **21**, 283–292.

Galzin, A-M., Loo, H., Sechter, D., *et al* (1986) Lack of seasonal variation in platelet [^{3}H]imipramine binding in humans. *Biological Psychiatry*, **21**, 876–882.

Gentsch, C., Lichtsteiner, M., Gaspar, M., *et al* (1985) ^{3}H-imipramine binding sites in platelets of hospitalised psychiatric patients. *Psychiatry Research*, **14**, 177–187.

Hamilton, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–296.

Healy, D., Whitehouse, A. M., White, W., *et al* (1990) Platelet α_2-adrenoceptor and ^{3}H-imipramine binding during desipramine administration and withdrawal in healthy volunteers. *Neuropsychopharmacology* (in press).

Hrdina, P. D., Lapierre, Y. D., Horn, E. R., *et al* (1985) Platelet ^{3}H imipramine binding: a possible predictor of response to antidepressant treatment. *Progress in Neuropsychopharmacology and Biological Psychiatry*, **9**, 619–623.

Innis, R. B., Charney, D. S. & Heninger, G. R. (1987) Differential ^{3}H-imipramine platelet binding in patients with panic disorder and depression. *Psychiatry Research*, **21**, 33–41.

Kanof, P. D., Coccaro, E. F., Johns, C. A., *et al* (1987) Platelet [^{3}H]imipramine binding in psychiatric disorders. *Biological Psychiatry*, **22**, 278–286.

Langer, S. Z., Zarifian, E., Briley, M., *et al* (1982) High-affinity ^{3}H-imipramine binding: a new biological marker in depression. *Pharmacopsychiatry*, **15**, 4–10.

Lewis, D. A. & McChesney, C. (1985) Tritiated imipramine binding distinguishes amongst subtypes of depression. *Archives of General Psychiatry*, **42**, 485–488.

Lowry, O. H., Rosebrough, N. J., Farr, A. L., *et al* (1951) Protein measurement with the Folin phenol reagent. *Journal of Biological Chemistry*, **193**, 265–275.

Maj, M., Mastronardi, P., Cerreta, A., *et al* (1988) Changes in platelet ^{3}3H-imipramine binding in depressed patients receiving electroconvulsive therapy. *Biological Psychiatry*, **24**, 469–472.

Mellerup, E. T., Plenge, P. & Rosenberg, R. (1982) ^{3}H-imipramine binding sites in platelets from psychiatric patients. *Psychiatry Research*, **7**, 221–227.

—— & Plenge, P. (1988) Imipramine binding in depression and other psychiatric disorders. *Acta Psychiatrica Scandinavica*, **78** (suppl. 345), 61–68.

Nemeroff, C. B., Knight, D. L., Krishnan, R. R., *et al* (1988) Marked reduction in the number of platelet-tritiated imipramine binding sites in geriatric depression. *Archives of General Psychiatry*, **45**, 919–923.

PAUL, S. M., REHAVI, M., SKOLNICK, P., *et al* (1981) Depressed patients have decreased binding of tritiated imipramine to platelet serotonin "transporter". *Archives of General Psychiatry*, **38**, 1315–1317.

PAYKEL, E. S. (1985) The clinical interview for depression. Development, reliability and validity. *Journal of Affective Disorders*, **9**, 85–96.

PLENGE, P., MELLERUP, E. T. & GJERRIS, A. (1988) Imipramine binding in depressed patients diagnosed according to different criteria. *Acta Psychiatrica Scandinavica*, **78**, 156–161.

POIRIER, M-F., BENKELFAT, C., LOO, H., *et al* (1986) Reduced Bmax of [^{3}H]-imipramine binding to platelets of depressed patients free of previous medication with 5HT uptake inhibitors. *Psychopharmacology*, **89**, 456–461.

——, GALZIN, A-M., LOO, H., *et al* (1987) Changes in [^{3}H]5-HT uptake and [^{3}H] imipramine binding in platelets after chlorimipramine in healthy volunteers. Comparison with maprotiline and amineptine. *Biological Psychiatry*, **22**, 287–302.

RAISMAN, R., SECHTER, D., BRILEY, M. S., *et al* (1981) High-affinity ^{3}H-imipramine binding in platelets from untreated and treated depressed patients compared to healthy volunteers. *Psychopharmacology*, **75**, 368–371.

——, BRILEY, M. S., BOUCHAMI, F., *et al* (1982) ^{3}H-Imipramine binding and serotonin uptake in platelets from untreated depressed patients and control volunteers. *Psychopharmacology*, **77**, 332–335.

ROY, A., EVERETT, D., PICKAR, D., *et al* (1987) Platelet tritiated imipramine binding and serotonin uptake in depressed patients and controls. Relationship to plasma cortisol levels before and after dexamethasone administration. *Archives of General Psychiatry*, **44**, 320–327.

STATISTICAL ANALYTICAL SYSTEMS (1982) *SAS User's Guide: Basic Statistics*. Cary, USA: SAS Institute Inc.

SCHNEIDER, L. S., FREDRICKSON, E. R., SEVERSON, J. A., *et al* (1986) ^{3}H-Imipramine binding in depressed elderly: relationship to family history and clinical response. *Psychiatry Research*, **19**, 257–266.

SPITZER, R. L. & ENDICOTT, J. (1978) *Research Diagnostic Criteria for a Selected Group of Functional Disorders* (3rd edn). New York: New York Psychiatric Institute.

SURANYI-CADOTTE, B. E., LAFAILLE, F., SCHWARTZ, G., *et al* (1985) Unchanged platelet ^{3}H-imipramine binding in normal subjects after imipramine administration. *Biological Psychiatry*, **20**, 1237–1240.

THEODOROU, A. E., KATONA, C. L. E., DAVIES, S. L., *et al* (1989) ^{3}H-Imipramine binding to freshly prepared platelet membranes in depression. *Psychiatry Research*, **29**, 87–103.

WAGNER, A., ABERG-WISTEDT, A., ASBERG, M., *et al* (1985) Lower ^{3}H-imipramine binding in platelets from untreated depressed patients compared to healthy controls. *Psychiatry Research*, **16**, 131–139.

——, ——, ——, *et al* (1987) Effects of antidepressant treatments on platelet tritiated imipramine binding in major depressive disorder. *Archives of General Psychiatry*, **44**, 870–877.

WHITAKER, P. M., WARSH, J. J., STACEY, H. C., *et al* (1984) Seasonal variation in platelet ^{3}H-imipramine binding: comparable values in control and depressed populations. *Psychiatry Research*, **11**, 127–131.

WORLD HEALTH ORGANIZATION (1977) *The International Classification of Diseases* (9th edn). Geneva: WHO.

WING, J. K., COOPER, J. E. & SARTORIUS, N. S. (1974) *Measurement and Classification of Psychiatric Symptoms*. Cambridge. Cambridge University Press

David Healy, MD, MRCPsych, *Clinical Research Associate, Department of Psychiatry, University of Cambridge*; Andreas E. Theodorou, PhD, *Research Fellow, Department of Pharmacology and Clinical Pharmacology, St George's Hospital Medical School, London*; Andrew M. Whitehouse, MB, MPhil, MRCPsych, *Clinical Lecturer, Department of Psychiatry, University of Cambridge*; Kevin M. Lawrence, MSc, *Research Technician, Department of Pharmacology and Clinical Pharmacology, St George's Hospital Medical School, London*; William White, *Research Nurse, Department of Psychiatry, University of Cambridge*; Helen Wilton-Cox, FIMLS, *Research Technician, Department of Psychiatry, University of Cambridge*; Sally M. Kerry, MSc, *Statistician, Department of Pharmacology and Clinical Pharmacology, St George's Hospital Medical School, London*; *Roger W. Horton, PhD, *Senior Lecturer, Department of Pharmacology and Clinical Pharmacology, St George's Hospital Medical School, London SW17 0RE*; Eugene S. Paykel, MD, FRCP, FRCPsych, *Professor of Psychiatry, Department of Psychiatry, University of Cambridge*

*Correspondence

British Journal of Psychiatry (1990), 157, 216–220

Multimodal Imaging in Alzheimer's Disease

The Relationship between MRI, SPECT, Cognitive and Pathological Changes

J. A. O. BESSON, J. R. CRAWFORD, D. M. PARKER, K. P. EBMEIER, P. V. BEST, H. G. GEMMELL, P. F. SHARP and F. W. SMITH

Patients with a clinical diagnosis of Alzheimer's disease were studied using MRI, SPECT, and psychometric tests. Significant correlations between focal perfusion deficits and focal cognitive deficits were found. Significant correlations between regional relaxation time of white matter and psychometric tests of diffuse and focal categories were also found. Pathological examination confirmed Alzheimer's disease as the only diagnosis.

Proton magnetic resonance imaging (MRI) employs the reconstruction of data obtained when mobile protons of a tissue are excited by the application of an oscillating magnetic field in the radio-frequency range, to display an image of that data. An important measure obtained during this process is the spin–lattice relaxation time (*T*1), the time taken for the nuclei to return to equilibrium after excitation. Most protons available for excitation in tissue are those of water and the *T*1 can therefore be altered by changes in the free : bound ratio of water or water content (Mathur de Vre, 1984).

Using MRI, significant increases have been found in the white-matter *T*1 in patients with dementia compared with normal controls. This has been a consistent finding at magnetic field strengths of both 0.04T and 0.08T (Besson *et al*, 1985; Ebmeier *et al*, 1987). Furthermore, the *T*1 changes correlate with severity of the dementia. Selecting specifically patients with Alzheimer's disease (AD), *T*1 increases have been confirmed in both studies. Cerebral blood flow measured using carbon-15-labelled CO_2 and positron emission tomography (PET) has been shown to be reduced in AD (Frackoviack *et al*, 1981), and such reduction has also been observed using single-photon emission computerised tomography (SPECT) with iodine-132-labelled iodoamphetamine (Gemmell *et al*, 1984; Sharp, 1986) and more recently with 99m-Tc-hexamethylpropyleneamineoxime (HMPAO) (Gemmell *et al*, 1987).

The aims of the current study were:

(a) to determine whether regional changes in white-matter *T*1 bear a relationship to focal cognitive deficits measured by psychometric tests in patients with AD

(b) to determine whether cortical perfusion as measured by HMPAO and SPECT bears a relationship to cognitive deficits in patients with AD

(c) to determine whether the *T*1 values in the white matter are related to perfusion changes in the adjoining grey matter

(d) to explain what these changes might mean with reference to pathological examination of the patients who have so far come to autopsy.

Method

Twenty-one right-handed patients (mean age 69, range 57–76 years, 3 men) with a clinical diagnosis of probable AD were studied with MRI, but only the last 13 of these with HMPAO and SPECT (when the technique became available). Diagnostic criteria applied were those of Glen & Christie (1979). In essence, this was progressive dementia in the absence of space-occupying, infective and other neurological disease. Biochemical, haematological and endocrinological abnormalities and vascular disease were also excluded. With respect to the last item, patients who scored greater than four on the Hachinski Ischaemic Scale (Hackinski *et al*, 1975) were excluded.

All patients received a battery of cognitive tests. The vocabulary and block-design subtests of the Wechsler Adult Intelligence Scale (WAIS) were used to give an indication of general intellectual functioning (Weschler, 1955; Saville 1971). Verbal and visuospatial memory were assessed using the digit span (digits forwards and backwards) subtest of the WAIS, the visual reproduction (visual recall) and logical memory subtests from the Wechsler Memory Scale (Wechsler, 1945), the Munn test of facial learning (Munn, 1961), an additional test of recognition of famous faces, and the Corsi Block Tapping Test (Milner, 1971). Complex visual-perceptual processing was assessed using the Benton Facial Recognition Test (Benton *et al*, 1983) and the Raven's Coloured Progressive Matrices (Raven, 1965). Verbal fluency was tested using a controlled oral word-association test (Benton, 1968). Imaging was carried out using the Aberdeen 0.08 T 3.4 MHz resistive system (intercalated SR–IR pulse sequence, TR 1000 ms, TE 200 ms (Redpath *et al*, 1987)). SPECT imaging was carried out using an IGE400AT rotating gamma camera digitally interfaced to a Link Analytical MAPS 5050 data-acquisition system

15 minutes following intravenous injection of 750mBq Tc-complexed-HMPAO (Sharp *et al*, 1987).

In the case of MRI, T_1 determinations in the frontal, parietal and temporal regions were carried out on transverse sections 1 cm apart using the three midventricular-cortical sections. Regional T_1 was measured in white matter using the VAX computer system in which a standard area was plotted on each modality of the displayed images, T_1, inversion-recovery and proton density, avoiding contamination with cerebrospinal fluid (CSF) on the ventricular side and grey matter on the cortical side. The standard areas were selected on the basis of the measured volume on the most atrophic image. Identical volumes were measured in each patient and the means of three consecutive images were used in each calculation. With regard to measuring the perfusion on the HMPAO imaging, the corresponding three sections of the HMPAO images were examined using visual rating of the colour images for each corresponding region as described by Gemmell *et al* (1987). MRI and SPECT images are on different matrices. Computerised interpolation of images can be achieved by adjusting for their differing centres of gravity, rotational planes and scales. The mean of three blind raters was used to score the severity of the perfusion deficit. A four-point scale was used: normal, mild hypoperfusion, moderate hypoperfusion, and severe hypoperfusion. The raters had a high inter-rater reliability ($r = 0.90$, $P < 0.001$). This system has previously been successfully used (Reid *et al*, 1988). Autopsies were carried out on six of the patients and the brains were examined after fixation in formalin.

Results

Cognitive test scores and regional white-matter T_1

The white-matter T_1 (MS) values in patients with AD were: for the right frontal region, mean 314 (range 293–360); left frontal region, mean 310 (range 290–358); right parietal region, mean 343 (range 301–398); left parietal region, mean 341 (range 297–395); right temporal region, mean 321 (range 303–338); and left temporal region, mean 321 range 301–340).

The results for the correlations between regional T_1 and the scores for the logical memory and delayed recall of logical memory showed a similar profile (Table I). There was a significant correlation with both left and right frontal and parietal, and left temporal white-matter T_1. This was particularly strong in the frontal region and more so on the left than on the right. The result is in keeping with the fact that this test, which demands attention and organisation as well as memory, reflects diffuse and widespread hemispheric damage, and since it is a verbal task, particularly on the left. A similar pattern of correlation was observed between the visual recall and delayed visual recall subtests and white-matter T_1. Delayed visual recall showed a stronger correlation overall and, in keeping with the visuospatial nature of this test, a strong relationship with white-matter change in right-sided structures.

The score on the vocabulary subtest of the WAIS (Table I) showed a higher correlation with the T_1 of left-sided structures compared with right, and for the more posterior

TABLE I

Correlations (Pearson's coefficient) between regional T_1 white matter and cognitive test scores

Region	*Logical memory*[1]	*Logical memory (delayed)*[1]	*Visual recall*[1]	*Visual recall (delayed)*[1]	*Vocabulary*[2]	*Munn facial learning*	*Verbal fluency*[3]	*Benton facial recognition*	*Corsi block tapping*	*Famous faces recognition*
Right frontal	0.51**	0.60**	0.54**	0.71***	NS	NS	NS	NS	NS	NS
Left frontal	0.60*	0.67**	0.47*	0.71***	NS	NS	0.42*	NS	NS	NS
Right posterior parietal	0.40*	0.62**	0.50*	0.66**	0.49*	0.61*	NS	0.80**	0.60**	NS
Left posterior parietal	0.50*	0.56**	0.44*	0.57**	0.52**	0.43*	NS	0.76**	NS	NS
Right temporal	NS	NS	0.57**	0.60**	NS	NS	NS	NS	NS	NS
Left temporal	0.40*	0.59**	NS	NS	0.44*	NS	NS	NS	NS	NS

*$P<0.05$, **$P<0.01$, ***$P<0.001$, 1. Subtest of Wechsler Memory Scale. 2 Subtest of WAIS. 3. Controlled oral word-association test (Benton, 1968).

locations (i.e. parietal and temporal). This is in keeping with the expected findings of vocabulary impairment in diffuse left-hemisphere disorder. Scores on both the Munn test of recognition of recently presented novel faces and the Benton Facial Recognition Test correlated with the parietal white-matter change more strongly on the right than on the left, as expected (Damasio *et al*, 1986).

Scores on the Corsi Block Tapping Test correlated with right parietal white-matter change, an observation which supports the finding of De Renzi *et al* (1977) that lesions in this region seriously impair performance on this task. The verbal fluency test, where subjects must produce as many words as possible beginning with a selected letter of the alphabet within a specified time, bore the expected relationship to the left frontal white-matter T_1.

No significant correlations were found with: orientation (WAIS block-design and Raven's tests); digits forward, which is a left inferior parietal function (highly focal changes may not be detected in the relatively large volumes of tissue measured); and digits backwards, which is influenced by more diffuse and predominantly left-hemisphere disturbance.

Cognitive test score and regional grey-matter perfusion

With regard to the correlations between perfusion deficit and performance on cognitive tests (Table II), the digits forwards and digits backward subtest scores correlated predominantly with right-sided changes posteriorly and anteriorly. No such correlation was found between these test scores and white-matter T_1. The logical memory and delayed logical memory subtests did not show any correlation with cerebral perfusion. It is to be noted that there was a relationship with white-matter T_1. Scores on the Corsi Block Tapping Test show a significant correlation with right but not left posterior parietal perfusion. The block-design subtest of the WAIS also showed correlation with posterior right parietal perfusion. With regard to the Munn test of facial learning, the score showed a correlation with right parietal perfusion. Overall, the visuospatial tasks appear to correlate more highly with perfusion deficit than do auditory verbal tasks.

To summarise:

(a) the Corsi Block Tapping Test, Munn facial learning test, Benton Facial Recognition test, and vocabulary subtest of the WAIS all showed strong correlations with cerebral blood-flow deficits in the grey matter as well as increased T_1 in the white matter, predominantly in the parietal regions (the visual recall subtest just failed to reach significance with perfusion deficits)

(b) The digits forward and digits backwards subtest, the famous faces recognition test, the Raven's Coloured Progressive Matrices and the block-design subtest correlated with cerebral perfusion only

(c) the logical memory subtest of the Wechsler Memory Scale and the verbal fluency test correlated with white-matter T_1 only.

TABLE II

Correlations (Kendall's coefficient) between regional perfusion deficits and cognitive test scores

Region	*Digits forwards*[1]	*Digits backwards*[1]	*Vocabulary*[1]	*Logical memory*[2]	*Logical memory (delayed)*[2]	*Corsi block tapping*	*Block design*[1]	*Ravens colour matrices*	*Famous faces recognition*	*Munn facial learning*	*Visual recall*[2]	*Benton facial recognition*
Left frontal	−0.06	−0.28	−0.29	−0.06	−0.39	0.07	0.12	−0.18	−0.47*	−0.37	−0.24	0.57
Right frontal	−0.06	−0.38	−0.38	−0.04	−0.35	−0.01	−0.04	−0.20	−0.48*	−0.42*	−0.30	0.35
Left posterior parietal	−0.12	−0.21	−0.10	−0.14	−0.29	−0.14	−0.38	−0.26	0.06	0.13	−0.40	−0.68*
Right posterior parietal	−0.53*	−0.49*	−0.48*	−0.22	0.14	−0.50*	−0.58*	−0.49*	0.27	−0.47*	−0.40	−0.72*

*$P<0.05$. 1. Subtest of WAIS. 2. Subtest of Wechsler Memory Scale.

Correlations between perfusion deficits and adjacent white matter

If the perfusion score in the posterior parietal region is plotted against regional posterior parietal white-matter T_1 lying adjacent to that area of cortex, significant correlations are found (on the right, $r=0.46$, $P<0.03$; on the left $r=0.63$, $P<0.003$). The greater the T_1, the greater the perfusion deficit on the adjoining grey matter, and the lower the blood flow in the areas. When similar comparisons were made for other regions, no significant correlations were obtained.

Pathological findings

The brains of six patients studied by both MRI and SPECT were examined at autopsy both macroscopically and microscopically by the same neuropathologist (PVB) in accordance with the criteria laid down in the Report from the Medical Research Council's (1987) AD workshop. AD was confirmed as the only diagnosis. In none of the cases were there significant atheromatous changes in the major arteries or macroscopically identifiable infarcts. Microscopically, those areas of the brain that had shown reduced cerebral perfusion with HMPAO were not detectably different from areas that had not shown such change. Rarefaction in the central white matter was found in five of the six cases, but there was no macrophage infiltration or other acceptable evidence of ischaemic damage. Small arteries showed mild fibrous sclerosis in three cases, in one of which there was also mild amyloid angiopathy; in none of the affected bessels was the lumen significantly narrowed and neither old nor recent thrombosis was identified.

Discussion

The results suggest that, where the strongest correlations between one of the image characteristics (perfusion or T_1) and cognitive test score occurs, there also tends to be a significant correlation between the other image characteristic and that cognitive test score. Thus tests including the Benton Facial Recognition, the Munn facial learning, the Corsi Block Tapping and the vocabulary subtest of the WAIS all show correlations with T_1 and blood flow. It is also of interest that tests which place demands on specific areas of the brain (e.g. Corsi Block Tapping and facial memory tasks) tend to show relationships with both imaging techniques, unlike those reflecting diffuse damage. Such specific focal abnormalities are particularly located in the parietal regions. However, such relationships between white-matter T_1 and grey-matter perfusion have not been found when the other brain regions are considered. While it is possible that this may be an incidental finding, it is noted that perfusion deficits in this region have been identified by a number of studies as a hallmark of AD (Risberg & Gustafson, 1983; Gemmell *et al*, 1984). It is interesting that the perfusion score in the parietal cortex correlates with the T_1 of the adjacent white matter on both left and right. It might be argued on the basis of the above data that the disruption of cognitive processes which require the support of mechanisms focused in specific cortical regions can be detected by changes in both regional T_1 and cerebral perfusion. However, failure on tasks which require a wide network of neural interconnections may be indicated only by degenerative changes in the white matter (which carries the brain communication tracts); then changes would only be detected by the T_1 index. Tasks on the verbal fluency test, finding the appropriate definition for a word or constructing a schema to remember a short sequence of events for example, are all extremely complex activities which require the integrity of a number of specialised processors. Integrity of the brain's inter-regional connections may be particularly important for the adequate performance of these tasks.

The histological appearances in the six brains so far examined suggest that the increased T_1 values in the white matter are not associated with ischaemia resulting from vascular disease. It seems that the rarefaction in the central white matter encountered in most of these cases (Besson *et al*, 1988) is more likely to be secondary to changes in the grey matter than to result from ischaemia (Englund *et al*, 1988). It is possible that axonal changes associated with nerve-cell degeneration may give rise to an increased water content in the white matter. The elevated T_1 may therefore be a reflection of the raised free : bound ratio of water consequent upon the alteration in myelin structure. A further possible explanation is that fluid increase in glial cells or in the extracellular compartment occurs in areas with loss of nerve fibres.

Conclusion

There are links between regional changes in white-matter T_1 values in AD and cognitive deficits. These indices of morphological change are associated with functional change (as indexed by reduction in regional cerebral perfusion). The regions of reduced perfusion and the region of increased T_1 are not due to recognisable vascular disease. The relationships between T_1 change and pathological change may allow the *in-vivo* interpretation of T_1 on images of patients with AD. Furthermore, these can be linked to functional impairment as measured by perfusion deficits and cognitive deficits.

References

Benton, A. L. (1968) Differential behavioural effects in frontal lobe disease. *Neuropsychologia*, **6**, 53–68.

——, Hamilton, K., Voraly, N. R., *et al* (1983) *Contributions to Neuropsychological Assessment*. New York: Oxford University Press.

Besson, J. A. O., Corrigan, F. M., Foreman, E. I., *et al* (1985) NMR imaging in dementia. *British Journal of Psychiatry*, **146**, 31–35

——, Ebmeier, K. P., Best, P. V., *et al* (1988) Do white matter changes on MRI and CT differentiate vascular dementia from Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 318–9.

Brun, A. & Englund, W. (1987) Brain changes in dementia of Alzheimer's type relevant to new imaging diagnostic methods. Progress in *Neuro-Psychopharmacological and Biological Psychiatry*, **10**, 297–308.

Damasio, R., Damasio, H. & Tranel, D. (1986) Prosopagnosia: anatomic and physiologic aspects. In *Aspects of Face Processing* (eds H. D. Ellis, M. Jeeves, A. Young, *et al*), pp. 293–301. Berlin: Springer-Verlag.

De Renzi, E., Faglioni, P. & Previdi, P. (1977) Spatial memory and hemisphere focus of lesion. *Cortex*, **13**, 424–433.

Ebmeier, K. P., Besson, J. A. O., Crawford, J. R., *et al* (1987) Nuclear magnetic resonance imaging and single photon emission tomography with radioiodine labelled compounds in the diagnosis of dementia. *Acta Psychiatrica Scandinavica*, **75**, 549–556.

Englund, E., Brun, A. & Alling, C. (1988) White matter changes in dementia of Alzheimer's type: biochemical and neuropathological correlates. *Brain*, **111**, 1425–1439.

Frackowiack, R. S. J., Pozzilli, C., Legg, N. J., *et al* (1981) Regional cerebral oxygen supply and utilisation in dementia. *Brain*, **104**, 753–778.

Gemmell, H. G., Sharp, P. F., Evans, N. T. S., *et al* (1984) Single photon emission tomography with 132-I-isopropyl-amphetamines in Alzheimer's disease and multi-infarct dementia. *Lancet*, *ii*, 1348.

——, ——, Besson, J. A. O., *et al* (1987) Differential diagnosis in dementia using 99m Tc-HMPAO: a new cerebral blood flow agent. *Journal of Computer Assisted Tomography*, **11**, 398–402.

Glen, A. I. M. & Christie, J. E. (1979) Early diagnosis of Alzheimer's disease: working definitions for clinical and laboratory criteria in Alzheimer's disease. In *Early Recognition of Potentially Reversible Deficits* (eds A. I. M. Glen, & L. J. Whalley). Edinburgh: Churchill Livingstone.

Hackinski, N. C., Iliff, L. D., Zilkha, E., *et al* (1975) Cerebral blood flow in dementia. *Archives of Neurology*, **32**, 632–637.

Mathur, de Vre, R. (1984) Biomedical implications of the relaxation behaviour of water related to NMR images. *British Journal of Radiology*, **57**, 955–976.

Medical Research Council (1987) *Alzheimer's Disease Workshop*. London: MRC.

Milner, B. (1971) Interhemispheric differences in the localisation of psychological processes in man. *British Medical Bulletin*, **27**, 272–277.

Munn, N. L. (1961) *Psychology: The Fundamentals of Human Adjustment* (4th edn). London: Harrap.

Raven, J. C. (1965) *Guide to Using the Coloured Progressive Matrices*. London: H. K. Lewis

Redpath, T. W., Hutchinson, J. N. S., Eastwood, L. M., *et al* (1987) A low-field NMR imager for clinical use. *Journal of Physics and Engineering: Scientific Instruments*, **20**, 1228–1234.

Reid, I. C., Besson, J. A. O., Best, P. V., *et al* (1988) Imaging of cerebral blood flow markers in Huntington's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 1264–68.

Risberg, J. & Gustafson, R. (1983) 133-xenon cerebral blood flow in dementia and in neuropsychiatry research. In *Functional Radionuclide Imaging of the Brain* (ed P. Magistretti). New York: Raven Press.

Saville, P. (1971) *A British Supplement to the Manual of the Wechsler Adult Intelligence Scale*. Windsor: NFER–Nelson.

Sharp, P. F., Gemmell, G. H., Cherryman, G. R., *et al* (1986) The application of 132-I-labelled (IMP) isopropylamphetamine imaging to the study of dementia. *Journal of Nuclear Medicine*, **27**, 761–68.

Wechsler, D. (1945) A standardised memory scale for clinical use. *Journal of Psychology*, **19**, 87–95.

—— (1955) *Manual for the Wechsler Adult Intelligence Scale*. New York: Psychological Corporation.

*J. A. O. Besson, BSc, MB ChB, DPM, MRCPsych, *Senior Lecturer, Department of Mental Health, University of Aberdeen, Foresterhill, Aberdeen AB9 2ZD*; J. R. Crawford, BSc, MSc, PhD, *Research Fellow, Department of Mental Health*; D. M. Parker, BA, PhD, FBPsS, CPsych, *Senior Lecturer, Department of Psychology*; K. P. Ebmeier, MRCPsych, *Lecturer, Department of Mental Health, currently at MRC Brain Metabolism Unit, Royal Edinburgh Hospital, Morningside, Edinburgh*; P. V. Best, FRCPath, *Senior Lecturer, Department of Pathology*; H. G. Gemmell, BSc, MSc, PhD, *Lecturer in Bio-Medical Physics*; P. F. Sharp, BSc, PhD, *Senior Lecturer, Department of Bio-Medical Physics, University of Aberdeen*; F. W. Smith, MB ChB, DMRD, FFR, *Consultant Radiologist, Specialist in Nuclear Medicine, Aberdeen Royal Infirmary, Foresterhill, Aberdeen.*

*Correspondence

British Journal of Psychiatry (1990), 157, 221–227

Daughters of People with Dementia
Expressed Emotion, Strain and Coping

K. D. BLEDIN, B. MacCARTHY, L. KUIPERS and R. T. WOODS

Twenty-five women who were the primary carers for a parent with dementia were rated for expressed emotion (EE) and then divided into two groups: 11 with low-EE ratings and 14 with high-EE ratings. Although there were no differences between these groups in the levels of their parents' cognitive impairment, when such impairment was taken into account, high-EE subjects were found to report higher levels of strain and distress. More efficient coping strategies were reported by subjects who made fewer critical comments and more positive remarks. High-EE subjects more frequently had no living siblings, and were more likely to have had a respite break from caring. EE status was not associated with continuing care in the community at a nine-month follow-up.

The number of very old people in the British population is increasing rapidly. Because of the high prevalence of dementia in these age groups, the absolute number of people suffering from dementia may be expected to increase correspondingly. Current financial and political realities make it unlikely that statutory facilities and services will be able to meet the demands of this section of the population, and increasing numbers of people in the community are going to find themselves as the principal carers of a demented elderly relative or parent.

A recent review of research on the emotional burden experienced by caregivers of dementia sufferers (Morris *et al*, 1988) reported discrepancies in findings due partly, they suggested, to the diversity of responses to caregiving. They identified mediating variables which might influence the caregiver's emotional response. These include the meaning carers attribute to the situation, the quality of their relationship with the dementia sufferer, their coping strategies, and the availability of formal and informal support.

However, the interplay of levels of coping, strain and distress in carers in the community, and the relevance of these factors to the continuation or breakdown of care have not been adequately determined (Gilleard, 1984*a*, 1987). Gilleard's (1987) findings suggested that reducing carers' distress may increase their willingness to continue providing care, even without any reduction in the objective problems. Coping strategies and the quality of the past relationship may determine, at least in part, the degree of strain and distress, and thus the commitment to continue caring (Morris *et al*, 1988).

A fuller understanding is needed of the associations between strain, distress and coping, and qualitative features of the relationship between carer and dependant. One measure of the affective quality of the relationships within families is expressed emotion (EE).

EE refers to ratings of emotional aspects of speech and verbal behaviour, which are reliable indicators of subsequent poor outcome in schizophrenia (Kuipers & Bebbington, 1988). The ratings are made on the basis of subjects' tone of voice and the content of their speech while they talk about the patients during the Camberwell Family Interview (CFI; Leff & Vaughn, 1985). The components of EE are critical comments, hostility, emotional overinvolvement, warmth and positive remarks.

EE studies have generally focused on families with a member with schizophrenia (e.g. Vaughn & Leff, 1976) or depression (e.g. Hooley *et al*, 1986) as well as on parents of girls with eating disorders (e.g. Szmukler *et al*, 1985) and adults with learning difficulties (Greedharry, 1987). However, no published study has yet examined EE in the children of people with psychiatric difficulties; most have concentrated on their spouses and parents. More specifically, there has been no published study of EE in children of people with dementia. A study by Orford *et al* (1987) examined EE in key relatives of 12 demented patients as part of a broader study, but only two of these relatives were children of the patients. Whittick (1987) used a Patient Rejection Scale as an approximation to EE critical comments and hostility in a study of people with dementia, but she did not use the CFI, which has been used in all EE research to date. Similarly, Gilhooly & Whittick (1989) did not use the CFI or the usual rating criteria in their recent study of caregivers' critical comments.

To the extent that EE measures reflect usual patterns of family interaction (e.g. Miklowitz *et al*, 1984), they may be a valuable source of information about the relationship between, in this case, the carer and the demented dependant as well as about how families cope with the care of an elderly person. Low-EE relatives may cope more effectively with the objective stresses of caring and as a result experience less subjective strain and distress. They may thus be able to maintain the elderly person in the community for longer. Psychosocial intervention aimed at reducing EE in families may then be helpful in improving coping, and/or reducing strain and distress, and possibly in promoting continuing community care (cf. interventions described by Leff *et al* (1982), Tarrier *et al* (1988) and others for families of patients with a functional illness).

One aim of the present study was to describe the characteristics of a sample of women caring for elderly parents with dementia in terms of EE ratings, levels of reported strain, minor psychiatric morbidity, use of coping strategies, and support. A further aim was to investigate whether EE was related to continued care in the community at a nine-month follow-up. On the basis of evidence from previous research (Morris *et al*, 1988), it was predicted that carers showing higher levels of the EE measures of critical comments and/or hostility would report higher levels of strain and distress than less critical or less hostile carers. The literature relating to coping strategies and support among carers of demented relatives has been less consistent in its findings. Thus their associations with EE levels were examined in an exploratory rather than predictive fashion.

Method

Thirty daughters caring for a parent with dementia were contacted through local-authority day-centres, community psychiatric nurses, and various MIND, Age Concern and Alzheimer's Disease Society day-centres and supporters' groups. Three of the daughters contacted were unwilling to participate and a further two agreed to participate, but mutually convenient dates and times could not be found. Thus 25 women were included in the study. They were the primary carers (i.e. four or more contacts per week – cf. Gilleard, 1984*b*) for a parent with Alzheimer-type or multi-infarct dementia. The women were known to the services and/or had been in contact with one or more of the self-help groups in their area. As Whittick (1988) has pointed out, day care for the demented elderly is often only offered when it is thought that the carer is under great stress or can no longer cope without support. The present sample cannot, therefore, be regarded as wholly representative of the population of daughter-carers in the community.

The daughters were interviewed using a structured demographic and social information schedule specially devised for the study and the brief version of the CFI which was tape recorded. They reported on their parents' impairment using the Behaviour and Mood Disturbance (BMD) Scale (Greene *et al*, 1982) and the Behaviour Rating Scale (BRS) of the Clifton Assessment Procedures for the Elderly (CAPE; Pattie & Gilleard, 1979). They also rated themselves on a measure of coping strategies (MacCarthy & Brown, 1989), on the Relatives' Stress Scale (RSS; Greene *et al*, 1982), on the 30-item General Health Questionnaire (GHQ; Goldberg, 1978), and on the BMD Stress Scale (BMD-stress), devised by Woods and his colleagues (personal communication, 1987) for their recent study of coping by spouses of demented patients. Subjects were required to rate levels of stress experienced when the patient did or did not do each of the items rated in the BMD Scale. Finally, when the parents were available ($n = 20$), they were assessed using the Cognitive Assessment Scale (CAS) of the CAPE. Five of the 25 parents were not available for this at the time of the researcher's visit.

EE ratings were subsequently made by an experienced EE rater (LK) from the recorded CFI interviews. The sample was divided into two groups on the basis of a median split of the EE 'critical comments' scores and the presence of rated 'hostility'. The two groups were then compared on a number of measures.

The daughters were followed up by telephone or postal questionnaire nine months after the initial interview, to ascertain whether or not their status as the primary carers had changed.

Results

One daughter refused to complete the self-report questionnaires. Thus some analyses are based on data from 24 respondents.

Demographic and social data

Daughters' ages ranged from 35 to 62 years (mean = 49.8, s.d. = 6.9). Nine (36%) were single, divorced or separated. Fourteen (56%) were in employment, more commonly full-time, and 17 (68%) were in social classes I–III (husbands or, if they were not married, themselves in professional, managerial, or skilled employment). Six (24%) had been the only child in the family; the great majority (80%) had no siblings living within 5 km of themselves, and a small majority (56%) had none within 32 km.

Twenty-one of the 25 parents were women. The average age of the parents was 82.4 years (s.d. = 6.1), and 12 (48%) were aged 85 or older (Table I). Three parents did not live with their daughters, and four daughters and parents had always lived together. Excluding these four, parents had been living with their daughters for a mean of 6.5 years (s.d. = 7.1), with a third co-resident for more than five years (Table I). Subjects had first noticed problems in the parents on average 4.7 years previously (s.d. = 2.9), and had had to look after them in a practical way for an average of 3.0 years (s.d. = 1.9).

All but three subjects (12%) had some social contact with their parent every day of the week and for 48% of the sample, contact was for more than eight hours per day. Forty per cent of parents never attended day-centres. Thus this sample comprised a high-contact group of carers

TABLE I
Background characteristics of the demented parents

Characteristic of demented parent	n	%
Age: years		
<75	3	12.0
75–84	10	40.0
⩾85	12	48.0
Period of co-residence: years		
not co-resident	3	12.0
<1	3	12.0
1–5	7	28.0
>5	8	32.0
always	4	16.0

and dependants. However, 66% of subjects had had a respite break during the period of caring, most commonly for one week, and for 56% this had been within the previous year. Only a small proportion of the subjects were readily willing to consider permanent institutionalisation for their parents, however: two women (8%) definitely and three (12%) possibly. A further 15 (60%) felt that they would consider this option only if the parent's problems made continued caring too difficult, and five (20%) would under no circumstances consider it.

EE results

The median critical comments score of four was used to define 'low' and 'high' EE. Eleven subjects were thus rated as low, with fewer than four critical comments in the hour-long interview, and the remaining 14 were rated as high with four or more critical comments. Nine of the daughters had hostility ratings greater than zero – i.e. they expressed at least *some* hostility towards the parents while talking about them (Table II). All of these nine subjects made four or more critical comments. The high-EE group was thus defined as: critical comments scores of four or more and/or hostility ratings greater than zero (Table II).

TABLE II
Expressed emotion (EE) component scores and ratings and frequency of face-to-face contact for low- and high-EE subjects

EE score/rating	*Low-EE subjects (n = 11)*		*High-EE subjects*[1] *(n = 14)*		*All subjects*	
	$\bar{x}$	*s.d.*	$\bar{x}$	*s.d.*	$\bar{x}$	*s.d.*
Critical comments	1.6	1.0	8.4	4.3	5.4	4.7
Positive remarks	0.8	1.5	1.4	1.7	1.2	1.6
Face-to-face contact: hours/week	64.7	32.2	49.0	18.9	55.9	26.2
	n	%	n	%	n	%
Hostility:						
absent (0)	11	100.0	5	35.7	16	64.0
present (>0)	0	0.0	9	64.3	9	36.0
Emotional overinvolvement:						
zero	7	63.6	9	64.3	16	64.0
one	4	36.4	5	35.7	9	36.0
Warmth:						
low (0–1)	2	18.2	7	50.0	9	36.0
high (2–4)	9	81.8	7	50.0	16	64.0

1. High-EE subjects: critical comment scores ⩾4 and/or hostility score >0.

A frequency count of the critical comments showed that types of behaviour most frequently criticised were not those which are often the least well tolerated by carers – incontinence, sleep disturbance, overdemanding behaviour, and the need for constant supervision (Morris *et al*, 1988). Instead, critical comments most frequently referred to repetitious speech and behaviour (15% of the total), messiness (11%), and aggressive argumentative behaviour (11%). Critical comments indicating disturbed sleep patterns constituted only three per cent of the total, as did those indicating overdemandingness and those concerning the caregivers' sense of being restricted by their role. Only one critical comment related directly to the parent's incontinence, but difficulties with toiletting were mentioned more often.

Emotional overinvolvement, which is an important EE feature in relatives of schizophrenic patients, was absent or low in the present sample. Only nine daughters (36%) were given a score greater than zero on this scale. Although carers reported that circumstances caused them to become very closely involved with the practical details of their parents' daily existence, this was rarely accompanied by a corresponding emotional overinvolvement. This measure was therefore not included in further analyses.

No subject was given the highest 'warmth' score, and about half of the sample made no 'positive remarks' about their parents during the interview. Face-to-face contact was high in this sample, with a mean of 55.9 hours per week, reflecting the physical and cognitive limitations

TABLE III
Social contact between daughters and parents with dementia: comparisons of low-EE and high-EE groups

	Low-EE subjects		*High-EE subjects*		*Fisher exact*
	n	%	n	%	P
Days/week at day-centre:					
0	2	18.2	8	57.1	<0.06
1+	9	81.8	6	42.9	
Respite care while primary carer:					
yes	5	45.5	12	85.7	<0.05
no	6	54.5	2	14.3	
Most recent respite care:					
<6 months ago	1	9.1	4	28.6	<0.10
⩾6 months ago	10	90.9	10	71.4	
Consider permanent institution?					
definitely not	4	36.4	1	7.1	<0.10
possibly/yes	7	63.6	13	92.9	

Table IV

Strain, distress and coping rated on a variety of scales: comparison of low-EE and high-EE groups

	Low EE			*High EE*			P
	n	$\bar{x}$	*s.d.*	n	$\bar{x}$	*s.d.*	
Strain/distress/coping variables							
RSS	11	23.2	10.1	13	28.9	9.4	$t=1.44$, 22 d.f. $P<0.10$
GHQ	11	4.8	7.0	13	9.4	6.3	$t=1.68$, 22 d.f. $P<0.10$
BMD-stress	11	46.4	30.6	13	36.2	19.7	NS
Duration of caring: months	11	34.4	24.9	14	36.6	22.7	NS
Positive coping[1]	11	39.2	5.1	13	41.4	6.4	NS
Maladaptive coping[1]	11	28.9	5.2	13	31.8	5.5	NS
Parent-impairment variables							
BMD	11	55.3	17.0	14	56.4	18.1	NS
CAPE: BRS	11	17.1	3.9	14	15.6	6.4	NS
CAPE: CAS	9	9.8	7.3	11	8.2	6.3	NS

[1] Rated on a measure of coping strategies (MacCarthy & Brown, 1989).

of the parents. For schizophrenic patients, 35 hours of face-to-face contact is regarded as the criterion cut-off in contributing to relapse (e.g. Vaughn & Leff, 1976).

The high- and low-EE groups were compared on each of the background variables. Six of the high-EE subjects had no siblings, as against none of the low-EE subjects, a significant difference (Fisher exact $P<0.02$). Significantly more high-EE subjects ($n=12$, 85.7%) than low-EE subjects ($n=5$, 45.5%) had had a respite break during their time as primary carer (Fisher exact $P<0.05$). The groups did not differ significantly from each other on any of the remaining demographic or social variables (Table III).

High-EE subjects tended to report more strain (RSS scores) and distress (GHQ scores) than low-EE subjects (Table IV). They scored over the threshold (4/5) for GHQ 'caseness' significantly more frequently than low-EE subjects (Fisher exact $P<0.02$). There were no significant differences between the high- and low-EE groups in their use of maladaptive or of positive coping strategies. Nor were any differences found between the groups on any of the measures of parent impairment (BMD and CAPE: BRS scores; Table IV).

Subjects who made one or more positive remarks ($n=13$) reported greater use of positive coping strategies than those who made no positive remarks ($n=12$) ($t=3.55$, 22 d.f., $P<0.005$). However, no significant differences were found between these two groups in reported strain, distress, or maladaptive coping, or in the parent-impairment variables. Similarly, no differences on these measures were found between subjects rated low in 'warmth' (EE ratings = 0–1; $n=9$) and those rated high in 'warmth' (EE ratings = 2–4; $n=16$).

Relationships among the coping, strain, distress and parent-impairment variables

Pearson product moment correlations showed that RSS and GHQ scores were highly correlated ($r=0.63$, $P<0.001$). Strain (RSS scores) in the relatives was positively associated with behavioural and mood distrubance in the parents ($r=0.56$, $P<0.005$ for BMD scores; $r=0.46$, $P<0.05$ for BRS scores); and with greater use of maladaptive coping strategies ($r=0.50$, $P<0.01$). However, maladaptive coping and GHQ scores were not significantly correlated, suggesting that use of maladaptive strategies was not simply a feature of the minor psychiatric morbidity assessed by the GHQ.

More maladaptive coping was associated with higher BMD scores ($r=0.39$, $P<0.05$). However, no significant correlation was found between positive coping scores and any of the subject-strain variables (RSS, GHQ and BMD-stress), nor between it and the parent-impairment variables (BMD, BRS, CAS).

Analyses of covariance were used to investigate possible interactions between the parent-impairment variables and the coping and subject-strain variables. Covariates in these analyses were the levels of impairment and/or disturbance of the parent (BMD, BRS, and CAS scores) and the length of time for which practical caring had been necessary. Since CAS scores were available for only 20 of the parents, separate analyses of covariance were performed, in which this variable served as the only covariate. In performing the analyses of covariance, the usual assumptions that the covariates were error-free were made.

When the parent's CAS score was the only covariate, high-EE subjects reported significantly higher levels of strain (RSS scores: $F(1,16)=8.51$, $P<0.01$) and distress (GHQ scores: $F(1,16)=10.55$, $P<0.005$) than low-EE subjects. However, there were no significant effects for either positive or maladaptive coping scores, or BMD-stress scores. Nor were any significant differences between the groups found when analyses were performed using BMD and BRS scores and the duration of caring as covariates.

Because of the apparent importance of the CAS scores in these analyses, the analyses were repeated on the smaller sample for which data were complete, with all three parent-impairment variables and duration as covariates. Once again, high-EE subjects were found to report higher levels of strain ($F(1,13)=15.86$, $P<0.005$) and distress ($F(1,13)=7.94$, $P<0.02$).

TABLE V
Daughters' status as carers at nine-month follow-up

Status as carer	*Low EE* n	*Low EE* %	*High EE* n	*High EE* %
No change	7	63.6	10	71.4
Change	1[1]	9.1	2[2]	14.3
Parent died	3	27.3	2	14.3

1. Parent in social services institution.
2. One parent moved to subject's son three months after interview; another on waiting list for social services institution.

Further analyses of covariance showed that subjects who made one or more positive remarks had higher positive coping scores than those who made no positive remarks. This finding held: (a) when the parent's CAS score was the only covariate ($F(1,16) = 6.28$, $P < 0.025$); (b) when covariates were BMD, BRS and duration of practical caring ($F(1,19) = 12.29$, $P < 0.05$); and (c) when all four independent variables were included in the analysis ($F(1,13) = 5.74$, $P < 0.05$). No further significant differences were found between these two groups. Nor did these analyses show any significant differences between subjects with higher and lower 'warmth' ratings.

Coping, EE and support

Maladaptive coping scores were higher among subjects making more critical comments, the main component of EE levels ($r = 0.45$, $P < 0.05$). They were also higher when less informal help was available ($r = -0.47$, $P < 0.02$), but formal support was more common among women who used more positive coping strategies ($r = 0.43$, $P < 0.05$).

Follow-up data

No significant difference was found between the high- and low-EE groups in the numbers whose status as primary carer had changed. Ten high-EE daughters (71%) and seven low-EE daughters (64%) continued to be the primary carers (Table V). Furthermore, carer status at follow-up was not associated with the ratings of warmth or number of positive remarks made during the CFI interviews.

Discussion

This is the first time that EE data have been described specifically for daughter-carers of demented parents. As in studies of relatives of patients with schizophrenia and depression (e.g. Vaughn & Leff, 1976), a wide range of frequencies of critical comments was found. However, the targets of these comments did not correspond closely with behaviour previously found to be particular sources of stress to carers, namely incontinence, sleep disturbance, and demanding behaviour (e.g. Gilleard, 1984*a*; Morris *et al*, 1988). Criticism seems to be evoked, rather, by behaviour which could be regarded as more deliberate and under the parent's control – repetition, messiness and argumentativeness. Incontinence, in contrast, although poorly tolerated, may be easier to recognise as being beyond the parent's active control.

Daughters who made at least four critical comments were highly likely also to express hostility towards their parents. Although the present sample was less critical, on average, than their counterparts living with schizophrenic or depressed relatives, it seems that if they were even mildly critical, these negative feelings generalised relatively easily into frank dislike or rejection. However, there were no high ratings on emotional overinvolvement, indicating: (a) that close involvement with the parents can often be interpreted as no more than the taking of realistic precautions; and/or (b) that one way to cope with the demands of caring for a demented parent is to maintain an emotional distance.

The latter contention was supported also by the low frequency of positive remarks. Some warmth was expressed by the majority of subjects, but ratings of this were generally low. During the interviews, even the most caring daughters described lost or altered relationships. In particular, they regretted the loss of reciprocity, and felt that their parents had changed irrevocably. This suggests that emotional distancing can occur from even a well-loved parent who has dementia, particularly, perhaps, when the carer adopts a 'care managing' rather than a 'care providing' strategy, as daughter-carers often do (Gilleard, 1984*a*).

The data provided considerable support for the hypotheses that lower levels of EE are associated with less strain and distress and with more efficient coping – more specifically, with less use of maladaptive coping strategies by those making fewer critical comments, and greater use of positive ones by those making any positive remarks. Whether a more positive attitude to the parent promotes more adaptive coping, or whether the use of positive coping strategies enables the maintenance of a more positive general approach cannot be ascertained from these cross-sectional data. However, these findings need to be pursued in further longitudinal research.

There were no differences between the high- and low-EE groups in the levels of impairment shown by their parents. However, when the level of the parents' cognitive impairment was taken into account, high-EE carers were shown to be experiencing significantly more strain and distress.

The high-EE subjects in this study were less likely than low-EE ones to have siblings. This finding raises questions about the nature of the relationship between parents and only children, and the extent to which early childhood patterns might persist into later life. An alternative, and simpler, explanation may be sought in terms of differences in the amount of informal support available to each group. However, no such difference was found in the present sample (Bledin, 1987).

High-EE subjects more frequently had respite breaks, and these tended to be more recent. The relevant services may have been more aware of tension in the high-EE families and thus been more active in facilitating respite arrangements. Alternatively, high-EE subjects may have been more willing to have their parents placed elsewhere, either temporarily or more permanently, since they were more open to the possibility of permanent institutionalisation for their parents. Low-EE subjects, by contrast, were more likely to have regular breaks from caring each week, when their parents attended day-centres (Table III).

The findings from this small sample suggest that EE levels are not associated, in the short term, with continuing care in the community. A longer interval between interview and follow-up might have been more revealing, but the mortality rate in this sample of elderly parents was, not surprisingly, high: 20% in nine months (Table V). A considerably larger sample would be needed to explore this question more fully.

In addition, the relevance of past and present intimacy (see Morris *et al*, 1988) to continuing community care remains to be established, and the relationship of these variables to the various EE measures also provides much scope for further research.

Conclusions

The present findings provide further information about the quality of the interactions between daughters and their demented parents which threaten the carers' well-being. Previous research has identified poor pre-morbid relationships, weak social support and a sense of losing control over their own reactions as risk factors for the carers (Morris *et al*, 1988). This study suggests that a critical attitude which, once present, often generalises to frank hostility, should be added to this list: carers expressing this emotional attitude towards their dementing parents were particularly likely to be experiencing strain and distress. As other studies have shown, this conjunction of negative attitudes and poor mental health occurs however disabled the parent is.

Thus interventions which aim to reduce strain and distress in carers by reducing their EE levels would seem to be a promising line to pursue. The illness process was often poorly understood, so that many of the high-EE daughters held unrealistic expectations about their parents' behaviour and tended to use unhelpful coping strategies to deal with everyday difficulties. A psychoeducational approach, combining education and family counselling, which has been used effectively with functionally ill patients and their families (eg. Leff *et al*, 1982; Tarrier *et al*, 1988) could therefore also be very relevant for this population.

Continued research is needed into the overlap between the key variables identified here and in other similar studies in order to optimise the quality of support offered to demented patients and their relatives while they continue to live together in the community.

Acknowledgements

Thanks are due to all those who assisted in the recruitment of subjects to this study. The first author was supported by grants from the Maudsley and Bethlem Royal Hospitals Special Health Authority Health Services Research Committee, and the Medical Research Council.

References

BLEDIN, K. D. (1987) Expressed emotion in daughters of people with dementia. Unpublished MPhil dissertation, University of London.

GILHOOLY, M. L. M. & WHITTICK, J. E. (1989) Expressed emotion in caregivers of the dementing elderly. *British Journal of Medical Psychology*, **62**, 265–272.

GILLEARD, C. J. (1984*a*) *Living with Dementia. Community Care of the Elderly Mentally Infirm*. London: Croom Helm.

—— (1984*b*) Problems posed for supporting relatives of geriatric and psychogeriatric day patients. *Acta Psychiatrica Scandinavica*, **70**, 198–208.

—— (1987) Influence of emotional distress among supporters on the outcome of psychogeriatric day care. *British Journal of Psychiatry*, **150**, 219–223.

GOLDBERG, D. P. (1978) *Manual for the General Health Questionnaire*. Windsor: NFER-Nelson.

GREEDHARRY, D. (1987) Expressed emotion in the families of the mentally handicapped: a pilot study. *British Journal of Psychiatry*, **150**, 400–402.

GREENE, J. G., SMITH, R., GARDINER, M., *et al* (1982) Measuring behavioural disturbance of elderly demented patients in the community and its effects on relatives: a factor analytic study. *Age and Ageing*, **11**, 121–126.

HOOLEY, J. M., ORLEY, J. & TEASDALE, J. D. (1986) Levels of expressed emotion and relapse in depressed patients. *British Journal of Psychiatry*, **148**, 642–647.

KUIPERS, L. & BEBBINGTON, P. (1988) Expressed emotion research in schizophrenia: theoretical and clinical implications. *Psychological Medicine*, **18**, 893–909.

LEFF, J. & VAUGHN, C. (1985) *Expressed Emotion in Families*. New York: Guilford Press.

——, KUIPERS, L., BERKOWITZ, R., *et al* (1982) A controlled trial of social intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, **141**, 121–134.

MacCarthy, B. & Brown, R. (1989) Psychosocial factors in Parkinson's disease. *British Journal of Clinical Psychology*, **28**, 41–52.

Miklowitz, D. J., Goldstein, M. J., Falloon, I. R. H., *et al* (1984) Interactional correlates of expressed emotion in the families of schizophrenics. *British Journal of Psychiatry*, **144**, 482–487.

Morris, R. G., Morris, L. W. & Britton, P. G. (1988) Factors affecting the emotional well-being of the caregivers of dementia sufferers. *British Journal of Psychiatry*, **153**, 147–156.

Orford, J., O'Reilly, P. & Goonatilleke, A. (1987) Expressed emotion and perceived family interaction in the key relatives of elderly patients with dementia. *Psychological Medicine*, **17**, 963–970.

Pattie, A. H. & Gilleard, C. J. (1979) *Manual of the Clifton Assessment Procedures for the Elderly (CAPE)*. Sevenoaks: Hodder & Stoughton.

Szmukler, G. I., Eisler, I., Russell, G. F. M., *et al* (1985) Anorexia nervosa, parental 'expressed emotion' and dropping out of treatment. *British Journal of Psychiatry*, **147**, 265–271.

Tarrier, N., Barrowclough, C., Vaughn, C., *et al* (1988) The community management of schizophrenia: a controlled trial of a behavioural intervention with families to reduce relapse. *British Journal of Psychiatry*, **153**, 532–542.

Vaughn, C. E. & Leff, J. P. (1976) The influence of family and social factors on the course of psychiatric illness: a comparison of schizophrenic and depressed neurotic patients. *British Journal of Psychiatry*, **129**, 125–137.

——, Snyder, K., Jones, S., *et al* (1984) Family factors in schizophrenic relapse: replication in California of British research in Expressed Emotion. *Archives of General Psychiatry*, **41**, 1169–1177.

Whittick, J. E. (1987) Expressed emotion and coping techniques among carers of the demented elderly. Unpublished paper presented to the Annual Conference of Psychologists' Special Interest Group in the Elderly, Edinburgh.

—— (1988) Dementia and mental handicap: emotional distress in carers. *British Journal of Clinical Psychology*, **27**, 167–172.

*K. D. Bledin, BA, MSc, PhD, MPhil, *Senior Clinical Psychologist, Redbridge Health Authority, District Psychology Service, Goodmayes Hospital, Barley Lane, Ilford, Essex IG3 8XJ*; B. MacCarthy, BA, MSc, Dip Clin Psych, *Lecturer in Psychology, Academic Unit, St Bernard's Wing, Ealing Hospital, Uxbridge Road, Middlesex*; L. Kuipers, BSc, MSc, PhD, *Honorary Principal Clinical Psychologist, The Maudsley Hospital, Denmark Hill, London, and Lecturer in Clinical Psychology, Institute of Psychiatry, London SE5 8AF*; R. T. Woods, MA, MSc, *Senior Lecturer in Clinical Psychology, Institute of Psychiatry, London SE5 8AF*

*Correspondence

British Journal of Psychiatry 1990, **157**, 228–231

Further Change in the Pattern of Mental Illness in the Elderly

ALEX B. CHRISTIE and ERIC R. M. WOOD

The methods of Roth's 1948–49 Graylingwell study, previously applied to a Crichton cohort of elderly patients of the years 1974–76, were repeated in 1984–86. The results support the continuing validity of Roth's classification of mental illness in the elderly, but changes in the diagnostic distribution of the cohort, previously observed in the 1970s study, are further in evidence in the 1980s one. Admissions of dementia cases continued to increase both relatively and absolutely, while the trend of increasing survival in dementia also continued.

The work of Roth (1955) in his Graylingwell study is a well known landmark in old age psychiatry. In this he classified a group of patients over 60 years of age admitted to Graylingwell Hospital in the years 1948–1949 into five principal diagnostic subgroups: affective psychoses; late paraphrenia; senile psychosis (now more commonly referred to as senile dementia of the Alzheimer type (SDAT)); arteriosclerotic dementia (now more commonly described as multi-infarct dementia (MID)); and acute confusional states. At follow-up at six and 24 months he demonstrated important differences in outcome between the respective diagnostic groups, and offered these as support for his classification. The nosology developed in Roth's study and its linkage with prognosis have provided the foundation for a number of subsequent comparative studies in Nottingham (Shah *et al*, 1969), Toronto (Duckworth *et al*, 1979), Newcastle (Blessed & Wilson, 1982) and Crichton (Christie, 1982). The results of these reflect continuing social, clinical and demographic changes. The present report seeks to develop what has already been established by employing a three-point comparison using Roth's original 1948–49 cohort, the 1974–76 Crichton cohort (Christie, 1982) as well as a more recent 1984–86 cohort from the same hospital.

Method

One minor methodological problem had to be overcome. Intake into Roth's study started at the age of 60 while our department accepted only cases over 64. Fortunately, the Graylingwell results were expressed in decades, and age-comparability was achieved by excluding both the age group of 60–69 years from Roth's original study and that of 65–69 years in the Crichton admissions, as Christie (1982) had done in the earlier study.

Accordingly, the diagnostic criteria of Roth's original study were applied to all patients over 69 years admitted to the psychogeriatric department of Crichton Royal Hospital in the years 1984–86. The patients were classified at six and 24 months as discharged, in-patient or dead. Results from this cohort were compared with patients of a similar age group in the 1948–49 Graylingwell and the 1974–76 Crichton Royal cohorts, with a view to identifying change in psychogeriatric experience over an extended period.

Results

A total of 321 cases were admitted in the three-year period. Six cases were lost to follow-up and a further 15 (5%) failed to fit into one of the five diagnostic categories. The exclusion rate of 5% is broadly comparable with that of 10% recorded by the studies at Crichton (Christie, 1982) and Newcastle (Blessed & Wilson, 1982). Table I shows the diagnostic distribution of all three cohorts.

TABLE I
Diagnostic distribution of the three cohorts

Diagnostic category	*Cohort*[1] *Graylingwell 1948–49* n *(%)*	*Crichton 1974–76* n *(%)*	*Crichton 1984–86* n *(%)*
Functional psychosis			
Affective psychosis	43 (30)	64 (24)	45 (15)
Late paraphrenia	12 (8)	14 (5)	4 (1)
Total functional	55 (38)	78 (29)	49 (16)
Dementia			
SDAT	64 (45)	100 (38)	193 (64)
MID	12 (8)	32 (12)	35 (12)
Total dementia	76 (53)	132 (50)	228 (76)
Acute confusional states	12 (8)	55 (21)	23 (8)
Grand Total	143 (100)	265 (100)	300 (100)

1. Figures in parenthesis are pecentage share of each cohort.

TABLE II
Outcome as percentages of each cohort at six and 24 months

	Outcome					
	Six months			*24 months*		
	Discharged %	*In-patient* %	*Dead* %	*Discharged* %	*In-patient* %	*Dead* %
Functional psychosis						
Affective psychosis						
Graylingwell 1948–49 (n=43)	53	35	12	42	19	40
Crichton 1974–76 (n=64)	75	9	16	70	6	23
Crichton 1984–86 (n=45)	84	4	11	67	2	31
Late paraphrenia						
Graylingwell 1948–49 (n=12)	8	75	17	9	45	45
Crichton 1974–76 (n=14)	93	7	–	64	7	29
Crichton 1984–86 (n=4)	75	25	–	50	50	–
Total functional						
Graylingwell 1948–49 (n=55)	44	44	12	35	25	40
Crichton 1974–76 (n=78)	78	9	13	69	6	24
Crichton 1984–86 (n=49)	84	6	10	65	6	29
Dementia						
SDAT						
Graylingwell 1948–49 (n=64)	13	25	63	5	8	88
Crichton 1974–76 (n=100)	28	57	15	15	35	50
Crichton 1984–86 (n=193)	29	62	9	19	46	35
MID						
Graylingwell 1948–49 (n=12)	17	33	50	7	7	87
Crichton 1974–76 (n=32)	25	19	56	14	32	55
Crichton 1984–86 (n=35)	23	51	26	9	31	60
Total dementias						
Graylingwell 1948–49 (n=76)	13	26	61	7	7	87
Crichton 1974–76 (n=132)	27	48	27	14	32	55
Crichton 1984–86 (n=228)	28	61	11	18	43	39
Acute confusional states						
Graylingwell 1948–49 (n=12)	58	17	25	67	–	33
Crichton 1974–76 (n=55)	62	13	25	47	4	49
Crichton 1984–86 (n=23)	91	–	9	65	–	35
Grand total						
Graylingwell 1948–49 (n=143)	29	32	39	23	13	65
Crichton 1974–76 (n=265)	49	29	22	37	18	45
Crichton 1984–86 (n=300)	42	47	11	29	34	37

In the 1984–86 cohort, affective psychoses accounted for only 15% of admissions compared with 24% in the earlier Crichton study and 30% in the Graylingwell cohort. Cases of late paraphrenia dropped almost to extinction. As a result, the percentage of patients with a functional psychosis fell from 38% in the Graylingwell study to 29% in the 1974–76 cohort and 16% in the 1984–86 one.

Among the dementias, a less consistent pattern emerges. In the earlier two cohorts, despite minor diagnostic variations, dementia accounted for 53% and 50% of cases respectively. In the 1984–86 period the figure rose to 76%. Admissions of toxic confusional states showed an increase in the 1970s but reverted to the 8% level in the 1980s.

Overall, a marked shift is shown from functional to organic admissions in old age psychiatry, and this took place despite a large increase in referrals across the diagnostic spectrum (Christie & Wood, 1987.) While the classification employed in official statistics renders precise comparison difficult, the evidence from national figures (statistics from Common Agency Scottish Health Service, 1975, 1984) suggests that the Crichton results reflect a broad pattern. A total of 43% of first admissions over 65 years old in 1975 were suffering from 'senile psychosis' compared with 51% with a similar diagnosis in 1984.

Table II records the outcome at six and 24 months for the three cohorts. For functional cases at six months, the discharge rate was marginally better in the 1984–86 study compared with the 1974–76 one, but both were vast improvements on that of the Graylingwell cohort. The death rates remained disappointingly constant at 10–13%.

Among the dementias, more marked changes were in evidence. The discharge rate rose from 13% in the 1940s study to 27% in the 1970s one but remained virtually unchanged at 28% in the 1980s. The death rate fell from 61% to 27% to 11%. These changes were mirrored by a corresponding increase in the numbers remaining in hospital (i.e. from 26% to 48% to 61%).

The numbers of acute confusional states varied but the broad pattern of outcome remained the same, namely discharge or death at six months with little in-patient burden resulting.

At the 24-month mark, the number of cases of functional psychosis in hospital had fallen, but the mortality rate for this group remained high at 29% in 1984–86, there being little change in the discharge rate since the 1970s.

More striking changes were in evidence with the dementia group. The discharge rate, having risen to 14% in the 1970s study, remained static in the 1980s one. However, the death rate continued to fall to less than half (39%) of that of the Graylingwell cohort (87%). The proportion of patients remaining in hospital rose from 7% to 32% to 42%.

The picture with acute confusional states was consistent with that recorded at six months.

Somewhat surprisingly, in view of demographic changes, the proportion of patients under 80 years remained remarkably constant between the 1974–76 (54%) and the 1984–86 (53%) study. These figures compare with a 78% share of the cohort noted in the same age group in the Graylingwell study.

Discussion

One must be impressed by the manner in which Roth's classification has withstood the test of time. In three recent studies – two at Crichton and one in Newcastle – not less than 90% of patients could be classified within his five original categories and, in the Crichton studies, a high proportion of the remainder consisted of 'graduates' with long established diagnoses such as schizophrenia.

Can we justify the claim that differences in outcome observed in the three studies, which were separated in time by 35 years and took place in two different hospitals, reflect broad evolutionary changes in the practice and experience of old-age psychiatry in Britain? Or are they merely of parochial interest – representing idiosyncratic differences of practice between hospitals and within a single hospital over a period of time?

We believe that we may assume more general relevance for three reasons. Firstly, a multicentre study in the 1950s (Royal Medico-Psychological Association, 1957), involving elderly patients from Graylingwell and Crichton Hospitals, showed very similar patterns of admission, diagnosis and outcome at that time. Secondly, results in the Newcastle study in the mid-1970s (Blessed & Wilson, 1982) parallel the Crichton experience at that time. Thirdly, the Crichton psychogeriatric service in the mid-1980s was compared in some detail with its operation ten years previously (Christie & Wood, 1987). Against a background of increased demand and demographic change, relatively minor changes in the style of practice or in available resources were revealed.

The impact of change in available resources and demand must be considered when evaluating the present findings. As previously reported (Christie & Wood, 1987), the referral rate rose by 150% over all diagnostic categories between 1974 and 1984 while resources remained virtually constant. A policy of earlier admission with consequent increase in survival appears unlikely. Dementia admissions in 1984 exhibited a similar degree of disability to their 1974 counterparts as measured by the Crichton Behavioural Rating Scale on admission.

Functional cases

Results in the affective group suggest that the introduction of antidepressant drugs in the 1960s may have had a profound effect on the prognosis in terms of improving discharge rates, but our evidence suggests that this process had run its course by the 1970s and that a major therapeutic advance is thus required to generate additional improvement.

Our methodology does not really allow us to engage in the debate on the prognosis of depression since broad outcomes such as discharge or in-patient status tell us little or nothing of the quality of life of the individual. We note, however, that the mortality rate remained high in the 1980s study and this inclines us towards the less optimistic view expressed by Post (1972) and Murphy (1983).

The downward trend in admissions of cases of late paraphrenia reported in the 1970s studies (Blessed & Wilson, 1982; Christie, 1982) continued into the 1980s despite the considerable increase in the number of referrals with this diagnosis between 1974 and 1984 (Christie & Wood, 1987). In the absence of any dramatic therapeutic breakthrough in the management of this condition, we attribute the change to improved community services and perhaps the high level of tolerance shown by our largely rural catchment area population.

Organic cases

As in the case of the late paraphrenias, we may dismiss the acute confusional states with two brief comments. Firstly, their fall in numbers from the 1970s to the 1980s probably reflects local developments in medical geriatric services as well as the view that such patients probably merit the full facilities of a district general hospital rather than a psychogeriatric admission ward. Secondly, the pattern of outcome remained clear-cut, i.e. discharge or death.

Homer *et al* (1988) have highlighted the problems of distinguishing between SDAT and MID when even the most sophisticated techniques are available. In an attempt to circumvent this problem we have reported our results as dementias without further discrimination on the assumption that, irrespective of errors in precise diagnosis, the numerical relationship between SDAT and MID remained reasonably constant between the 1970s and 1980s.

The relative and absolute increase in the number of cases of dementia from the 1970s to the 1980s is striking. This change cannot be explained simply on the basis of demographic change and the high prevalence of SDAT in older age groups since, as already stated, the proportion of patients above and below 80 years was almost constant in the first and second Crichton cohorts. The absence of an increase in the very elderly dementias in our more recent patient population is further evidence for the argument (Christie & Wood, 1988) that the characteristics of dementia in the very elderly differ from those in the younger age group, and, as a result, these cases may be managed in settings which, unlike psychogeriatric wards, do not require major behavioural difficulties as the prime criterion for admission.

The upward trend in survival from the 1940s to the 1970s, reported by studies employing the Roth methodology, continued in the 1980s. We can at this stage only advance the traditional explanations of improved services, more enlightened patient care, and the impact of modern drugs, particularly antibiotics, to account for the change.

Clearly, the present study can only indicate broad trends. However, whatever the explanations, the consequences of the changes reported here are important. Applying standardised rates of bed requirements at six and 24 months to the respective diagnostic groups, we find that cases of late paraphrenia have a minute requirement for long-stay placement. The overall requirement at six months for functional cases has continued to decline and appears, at two years, to have stabilised at six beds per 100 admissions. The dementias, on the other hand, pose increasing problems. The Graylingwell requirement at six months of 26 beds per 100 admissions rose in the 1970s Crichton study to 48 beds and in the 1980s one to 61. At two years, the figures were respectively 7, 32 and 43 beds per 100 admissions.

These results imply that to adopt norms for bed provision for dementia based on demographic factors alone would seriously underestimate the true requirement. The point has now been reached where lengthening patient survival must be incorporated into calculations of changing need.

References

BLESSED, G. & WILSON, I. D. (1982) The contemporary natural history of mental disorder in old age. *British Journal of Psychiatry*, 141, 59–67.

CHRISTIE, A. B. (1982) Changing patterns in mental illness in the elderly. *British Journal of Psychiatry*, 140, 154–159.

—— & WOOD, E. R M. (1987) Psychogeriatrics 1974 to 1984. expanding problems and fixed resources. *British Journal of Psychiatry*, 151, 813–817.

—— & —— (1988) Age, clinical features and prognosis in SDAT *International Journal of Geriatric Psychiatry*, 3, 63–68.

DUCKWORTH, G. S., KEDWARD, H. B., BAILEY, W. F. (1979) Prognosis in mental illness in old age *Canadian Journal of Psychiatry*, 24, 674–682.

HOMER, A. C., HONAVAR, M., LANTOS, P. L., *et al* (1988) Diagnosing dementia: do we get it right? *British Medical Journal*, 297, 894–896.

MURPHY, E. (1983) The prognosis of depression in old age. *British Journal of Psychiatry*, 142, 111–119.

POST, F. (1972) The management and nature of depressive illnesses in late life. A follow through study. *British Journal of Psychiatry*, 121, 392–404.

ROTH, M. (1955) The natural history of mental disorder in old age. *Journal of Mental Science*, 101, 281–301

ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION (1957) *The Course and Outcome of Mental Disorders in Old Age*. Report to the Council of the Royal Medico-Psychological Association by the geriatric sub-committee of the research and clinical section. London: RMPA.

COMMON SERVICES AGENCY SCOTTISH HEALTH SERVICE (1975) *Scottish Mental Hospital In-patient Statistics* (national comparison tables). Edinburgh: Information Services Division, CSASHS

—— (1984) *Scottish Mental Hospital In-patient Statistics* (national comparison tables). Edinburgh: Information Services Division, CSASHS.

SHAH, K. V., BANKS, G. D. & MERSKEY, H. (1969) Survival in atherosclerotic and senile dementia. *British Journal of Psychiatry*, 155, 1283–6.

*Alex B. Christie, MB, ChB, FRCP, FRCPsych, *Consultant Psychiatrist*; Eric R. M. Wood, MA, MB, BS, MRCPsych, *Consultant Psychiatrist, Department of Geriatric Psychiatry, Crichton Royal Hospital, Dumfries, Scotland*

*Correspondence

British Journal of Psychiatry (1990), 157, 232–238

A Double-Blind Placebo-Controlled Study of Buspirone in Diazepam Withdrawal in Chronic Benzodiazepine Users

C. H. ASHTON, M. D. RAWLINS and S. P. TYRER

A double-blind placebo-controlled trial of 23 chronic benzodiazepine users showed that overall, buspirone did not appear to be helpful in alleviating benzodiazepine withdrawal symptoms. Buspirone (5 mg t.d.s.) or placebo was administered for four weeks before, during and after diazepam withdrawal. Patients taking buspirone had a markedly higher dropout rate (seven out of 11) than those taking placebo (one out of 12). Mean daily diazepam dosage at entry was significantly higher in the buspirone group, but overall initial diazepam dosage was not related to outcome. Higher subjectively rated anxiety at the start of withdrawal was significantly related to higher dropout rate, irrespective of treatment, and was greater (although not significantly so) in the buspirone group.

Buspirone has recently been introduced as a new type of anxiolytic drug, with possible advantages over benzodiazepines (*The Lancet*, 1988). Anxiolytic efficacy comparable to that of benzodiazepines has been reported in some (Feighner *et al*, 1982; Rickels *et al*, 1982; Tyrer & Owen, 1984) but not all (Olijade & Lader, 1987*b*; Olijade, 1988) clinical studies. The mode of action of buspirone is not clear but it has been suggested that its anxiolytic activity is due to agonist effects at 5-hydroxtryptamine$_{1A}$ (5-HT$_{1A}$) receptors (Fozard, 1987; Dourish *et al*, 1986; Traber & Glaser, 1987). It does not bind to benzodiazepine or post-synaptic gamma-aminobutyric acid$_A$ (GABA$_A$) receptors (Taylor *et al*, 1980; Eison & Temple, 1986).

The present study reports a double-blind placebo-controlled trial of the utility of buspirone in benzodiazepine withdrawal. A previous trial (Olajide & Lader, 1987*a*) found that buspirone failed to suppress benzodiazepine withdrawal symptoms. However, the onset of action of buspirone is about three weeks (Murphy *et al*, 1989), and in the study of Olajide & Lader (1987*a*), the drug was administered only at the start of benzodiazepine withdrawal. In our investigation, designed to maximise any advantages of buspirone, the drug was given for four weeks before benzodiazepine withdrawal was commenced, and was continued throughout the four weeks of withdrawal and for a further four weeks after withdrawal.

Method

Patients

The patients were referred by their general practitioners (GPs) for help in benzodiazepine withdrawal. They were considered eligible for the trial if they were over 18, had been on continuous benzodiazepine therapy for a minimum of six months, wished to withdraw from benzodiazepines, were not taking other psychotropic medication nor abusing alcohol or drugs, and were free from major psychiatric or physical disease. Fully informed consent was obtained from both the patients and their GPs, and the trial was approved by the joint University/DHA Ethics Committee. A total of 23 patients entered the trial; their relevant characteristics are shown in Table I.

TABLE I

Characteristics of patients at start of trial in placebo or buspirone treatment groups

Patient characteristics	*Placebo (n = 12)*	*Buspirone (n = 11)*	*Difference*
Males : females (*n*)	6 : 6	3 : 8	NS
Mean age: years (s.d.)	43.4 (10.94)	39.8 (10.24)	NS
Pre-morbid personality disorder, yes : no (*n*)	5 : 7	2 : 9	NS
Mean duration of benzodiazepine usage: years (s.d.)	11.25 (4.47)	9.5 (5.60)	NS
Mean diazepam dosage at entry: mg/day (s.d.)	7.5 (4.6)	15.5 (8.2)	$t = 2.808$ $P < 0.05$
Mean plasma diazepam level at visit 3 (day 0): μ/l (s.d.)	208 (135)	434 (270)	$t = 2.235$ $P < 0.05$
Mean plasma nordiazepam level at visit 3 (day 0): μ/l (s.d.)	257 (222)	518 (330)	NS

Weeks		4	8	12	16
Days	–28	0	28	56	84
Visits	1 2	3 4	5 6 7 8	9 10	11 12 13
Placebo group	stabilise on diazepam syrup	diazepam syrup +placebo tablets	diazepam withdrawal +placebo tablets	placebo diazepam syrup +placebo tablets	placebo tablets
Buspirone group	stabilise on diazepam syrup	diazepam syrup +buspirone tablets	diazepam withdrawal +buspirone tablets	placebo diazepam syrup +buspirone tablets	placebo tablets

FIG. 1. Procedure for placebo-controlled trial of buspirone in diazepam withdrawal

Procedure

The patients were told that the trial would take 20 weeks and that at some time during this period their diazepam therapy would be withdrawn gradually. Those who were taking other benzodiazepines were changed to an equivalent dose of diazepam before the start of the study. This substitution caused no difficulties and did not increase symptoms. For all patients, the diazepam was administered in a liquid formulation labelled 'relaxing syrup', supplied by the hospital pharmacy at weekly or fortnightly intervals. The volume of syrup taken by the patients was constant throughout the trial, but the diazepam concentration was varied according to individual initial dosage and withdrawal requirements.

The study was carried out double blind. At entrance, the patients were randomly assigned to buspirone (11 patients) or placebo (12 patients) groups balanced for age, sex and pre-morbid personality disorder. Thereafter, treatment was administered in a series of four-week blocks as shown in Fig. 1. For the first four weeks all patients were maintained on their usual dose of diazepam, administered in the form of syrup. At the start of the second four weeks, either buspirone (5 mg t.d.s.) or matching placebo tablets were added to the constant dose of diazepam. During the third four weeks the diazepam was slowly withdrawn in both groups by reducing its concentration in the syrup by 25% each week until it was zero at the end of the fourth week. Patients then took placebo 'relaxing syrup' containing vehicle only. During the diazepam withdrawal period, patients also continued taking buspirone or placebo tablets.

For the fourth four weeks all patients continued on placebo 'relaxing syrup' and on either placebo or buspirone tablets. At the start of the fifth and final four weeks, the placebo 'relaxing syrup' was stopped and patients were told that they had successfully withdrawn from diazepam. At the same time, buspirone tablets were replaced with placebo tablets, and both groups continued to take placebo tablets for the last four weeks of the trial.

Clinical assessments

During the trial, patients attended for assessment on 13 occasions, spaced one or two weeks apart, as shown in Fig. 1. At entrance a full physical examination was carried out. Liver function tests, full blood count, serum electrolytes and urea and urinalysis were performed on visits 3, 9 and 13. Blood pressure and heart rate were measured at entrance and on visits 3, 9 and 13. Symptom assessments were recorded on each of the 13 visits and included the following four aspects.

Psychic
Drowsiness/fatigue
Excitability (jumpiness, restlessness)
Unreality
Poor memory/concentration
Perceptual distortion
Hallucinations
Obsessions
Agoraphobia/phobias
Panic attacks
Depression
Paranoid thoughts
Rage/aggression/irritability
Craving

Somatic
Headache
Pain (limbs, back, neck)
Pain (teeth, jaw)
Tingling, numbness, altered sensation (limbs, face, trunk)
Stiffness (limbs, back, jaw)
Weakness ('jelly legs')
Tremor
Muscles twitches
Ataxia
Dizziness/lightheadedness
Blurred/double vision

Somatic (continued)
Tinnitus
Speech difficulty
Hypersensitivity (light, sound, taste, smell)
Insomnia/nightmares
Fits
Nausea/vomiting
Abdominal pain
Diarrhoea/constipation
Appetite/weight change
Dry mouth
Metallic taste
Difficulty in swallowing
Flushing/sweating
Palpitations
Overbreathing
Thirst
Frequency/polyuria, pain on micturition
Incontinence
Menorrhagia, PMT
Mammary pain/swelling
Skin rash/itching
Stuffy nose/ sinusitis
Influenza-like symptoms
Sore Eyes
Other Symptoms (specify)

FIG. 2. Withdrawal symptoms rated on a scale of severity of 0 (none), 1 (mild), 2 (moderate), and 3 (severe).

(a) *Withdrawal symptoms.* A list of 49 symptoms (Fig. 2) selected from previous experience of benzodiazepine withdrawal (Ashton, 1984) were rated by the patients on a scale of severity (0 = none, 1 = mild, 2 = moderate, 3 = severe). Overall symptom scores were obtained from each visit by adding up the total of individual scores for each symptom listed.

(b) *Psychiatric rating scales.* These were chosen to provide a selection of observer and self-rating scales that measured anxiety and depression. The observer scales used were the Brief Scale for Anxiety (Tyrer *et al*, 1984), the Montgomery-Asberg Rating Scale for Depression (Montgomery & Asberg, 1979) and the Raskin-Covi Anxiety/Depression Scale (Raskin *et al*, 1969). The self-rating scale chosen was the Hospital Anxiety Depression Scale (HAD; Zigmond & Snaith, 1983), which comprises items specifically concerned with anxiety and depression. In addition a DSM-III (American Psychiatric Association, 1980) psychiatric diagnosis was made after interviewing the patient according to the Structured Clinical Interview for DSM-III (SCID; Spitzer *et al*, 1985), and the Personality Assessment Schedule (Tyrer & Alexander, 1979) was completed at entrance after interviewing a close informant of the patient.

(c) *Plasma diazepam and nordiazepam concentrations.* These were measured on visit 3 (start of diazepam withdrawal), visit 9 (end of withdrawal), visit 11 and visit 13.

(d) *Adjuvant medication.* Where indicated, 'escape' medication in the form of diphenhydramine for night sedation, paracetamol for pain, and propranolol for tremor and palpitations were prescribed. Patients were free to attend tranquilliser support groups if desired, and were able to drop out of the trial at any time. Patients who required further prescriptions of diazepam, outside the protocol, or antidepressant or other psychotropic drugs were withdrawn from the trial and counted as dropouts.

Results

A summary of the main findings is given in Tables I and II. At entrance to the trial there were no significant differences between the placebo and buspirone groups for age, sex, duration of benzodiazepine use, or personality disorder. However, despite the random assignment of patients, analysis revealed that the buspirone group were taking significantly higher daily doses of diazepam than the placebo group (mean daily dosage: placebo group 7.5 mg (s.d. 4.6); buspirone group 15.5 mg (s.d. 8.2); $t=2.808$, $P<0.05$). On visit 3, at the start of buspirone/placebo treatment, the buspirone group also had significantly higher plasma concentrations of diazepam (mean plasma diazepam concentration: placebo group 208 µg/l (s.d. 135); buspirone group 434 µg/l (s.d. 270); $t=2.235$, $P<0.05$). Plasma concentrations of nordiazepam were also higher in the buspirone group than in the placebo group, but the difference did not reach statistical significance. Plasma diazepam and nordiazepam concentrations correlated significantly with diazepam dosage (dosage versus diazepam concentration: $r=0.679$, $P<0.001$; dosage versus nordiazepam concentration: $r=0.751$, $P<0.001$).

In spite of the difference in diazepam dosage and plasma concentration, on visit 3 there were no significant differences between the groups in symptom score, Brief Scale for Anxiety (see Table II), Montgomery-Asberg Scale, and Raskin-Covi Depression Scale. The Raskin-Covi Anxiety score was significantly higher in the buspirone group (placebo group: 6.17 (s.d. 1.75); buspirone group: 8.09 (s.d. 2.59); $t=2.1064$, $P<0.05$), as was also the anxiety score on the HAD Scale (placebo group: 10.25 (s.d. 5.44); buspirone group: 15.18 (s.d. 4.60), $t=2.3345$, $P<0.05$).

However, at the start of benzodiazepine withdrawal (visit 5), there were no significant differences between groups on any of the assessment scales. In both groups, scores for all ratings rose during benzodiazepine withdrawal (visits 6–9) and continued to rise after withdrawal, reaching a peak on visit 10, two weeks after withdrawal. All except one of the 21 patients who underwent diazepam withdrawal showed an increase in symptoms and/or psychiatric ratings.

Mean withdrawal scores on nearly all scales were higher in the buspirone than in the placebo group, although the differences usually did not reach statistical significance until visit 10 (Brief Scale for Anxiety: placebo, 18.91 (s.d. 6.91), buspirone 27.33 (s.d. 8.98); $t=2.1668$, $P<0.05$;

TABLE II

Mean ratings for withdrawal symptoms and HAD (anxiety) in the two treatment groups

			Placebo			*Buspirone*			*Difference*	
Visit[1]	*(Day)*	n	$\bar{x}$	*s.d.*	n	$\bar{x}$	*s.d.*	t	*d.f.*	P
Withdrawal symptoms										
3	(0)	12	24.91	13.52	11	31.36	14.15			NS
5	(28)	12	28.33	18.86	9	29.33	17.52			NS
7[2]	(42)	12	34.00	22.86	9	37.11	18.27			NS
9[2]	(56)	12	27.58	17.96	8	33.87	13.35			NS
10	(70)	11	38.31	21.57	6	43.00	17.88			NS
11	(84)	11	35.00	20.80	5	41.40	17.42			NS
13	(112)	11	26.09	19.50	4	24.75	6.07			NS
HAD (anxiety)										
3	(0)	12	10.25	5.44	11	15.18	4.60	2.334	21	<0.05
5	(28)	11	9.09	5.50	9	13.11	4.45			NS
7[2]	(42)	12	9.41	5.45	9	15.0	2.95	2.093	19	<0.05
9[2]	(56)	11	9.18	6.07	8	14.25	4.68			NS
10	(70)	11	11.90	6.60	6	15.83	4.99			NS
11	(84)	11	10.18	6.72	4	13.25	7.18			NS
13	(112)	8	11.75	3.10	4	14.50	5.25			NS

1. Data from visits 1, 2, 4, 6, 8 and 12 are omitted for brevity.
2. Ratings during benzodiazepine withdrawal.

Montgomery-Asberg Scale: placebo 16.36 (s.d. 7.39), buspirone 24.66 (s.d. 6.37); $t=2.3138$, $P<0.05$; Raskin-Covi Anxiety Scale: placebo 7.09 (s.d. 2.07), buspirone 10.33 (s.d. 2.73); $t=2.7622$, $P<0.05$; HAD Scale (anxiety), visit 7: placebo 9.41 (s.d. 5.45), buspirone 15.00 (s.d. 2.95); $t=2.0937$, $P<0.05$). These differences occurred in spite of the fact that by visit 10 nearly half of the buspirone patients had dropped out of the trial (see below) and only those with relatively less severe symptoms remained. In those remaining, there was no evidence of a 'pseudowithdrawal' reaction when placebo diazepam syrup was stopped at visit 11.

In the placebo group, only one of the 12 patients failed to complete the trial. This subject dropped out after visit 9 following the sudden death of his father. By contrast, seven of the 11 patients in the buspirone group dropped out. Reasons for dropout included failure to attend (one patient), necessity for antidepressant medication (one patient), and unwillingness to continue because of severe anxiety (five patients). Two patients dropped out before diazepam withdrawal was commenced. The difference in dropout rate between the two groups was significant ($\chi^2=6.82$, $P<0.01$). The number of patients requiring added medication with propranolol or diphenhydramine after the start of diazepam withdrawal was not significantly different between the groups (three patients in the placebo group and four in the buspirone group). There was no evidence of a withdrawal reaction after stopping buspirone on visit 11, after 12 weeks of continuous use.

Outcome was significantly better in the placebo group than in the buspirone one. At six and 12 months after diazepam withdrawal, 11 of the 12 patients in the placebo group, but only six of the 11 buspirone patients, were still off benzodiazepines ($\chi^2=4.101$, $P<0.05$).

Thus, the buspirone group fared considerably worse than the placebo group with respect to withdrawal symptoms and psychiatric ratings, dropout rate, and ultimate success in stopping diazepam. Factors which might explain this discrepancy, including higher initial diazepam dosage, higher initial anxiety ratings, plasma concentrations, characteristics of buspirone itself and personality/psychiatric factors were considered.

Initial diazepam dosage

As shown on Table III, there was no difference in initial diazepam dosage (irrespective of treatment group) between those who achieved a successful outcome and those who did not, or between those who dropped out and those who did not. Moreover, there were no significant correlations between pre-withdrawal diazepam dosage and symptom scores at visit 10 (correlations: mean initial diazepam dosage v. symptom score, $r=0.274$ (NS); v. Brief Scale for Anxiety, $r=0.269$ (NS); v. Montgomery-Asberg Scale, $r=0.308$ (NS); v. Raskin-Covi Anxiety, $r=0.274$ (NS); v. Raskin-Covi Depression, $r=0.188$ (NS); v. HAD Scale (anxiety), $r=0.096$ (NS); and v. HAD Scale (depression), $r=0.002$ (NS).

It was noted that the higher the initial diazepam dosage, the greater the scores for anxiety and depression on the observer scales. However, the very low correlation on the self-rating scales suggest that high diazepam dosage in itself did not predispose to increased dysphoria after withdrawal.

Bearing in mind the general lack of relationship between initial diazepam dosage and successful outcome, dropout rate, and symptom scores, the poorer clinical course in the buspirone group could not be explained by the difference in initial diazepam dosage.

Initial anxiety scores

Scores on six of the seven ratings at the start of benzodiazepine withdrawal (visit 5) were not significantly

TABLE III

Relations between clinical course of diazepam withdrawal and pre-withdrawal diazepam dosage

Clinical course (independent of treatment)	n	*Mean pre-withdrawal diazepam dosage: mg/day (s.d.)*	*Difference*
Off diazepam at 6 and 12 months	17	11.2 (7.4)	t=0.128 NS
On diazepam at 6 and 12 months	6	11.7 (8.5)	
Dropouts	8	11.56 (5.8)	t=0.112 NS
Non-dropouts	15	11.17 (9.4)	

TABLE IV

Relations between clinical course of diazepam withdrawal and HAD (anxiety) scores at start of withdrawal (visit 5)

Clinical course (independent of treatment)	n	*Mean HAD (anxiety) scores at start of diazepam withdrawal (visit 5) (s.d.)*	*Difference*
Off diazepam at 6 and 12 months	15	9.40 (4.99)	t=2.44 P<0.05
On diazepam at 6 and 12 months	5	15.40 (3.78)	
Dropouts	8	15.17 (3.31)	t=2.70 P<0.05
Non-dropouts	15	9.07 (5.04)	

different (independent of treatment group) between those who achieved a successful outcome at six and 12 months and those who did not, nor between dropouts and non-dropouts. However, as shown in Table IV, scores on the HAD Scale (anxiety) at start of withdrawal were higher in those who failed to withdraw and in those who dropped out.

Similarly, for five of the seven assessment scales there were no significant correlations between pre- and post-withdrawal scores, irrespective of treatment group. However significant correlations were observed between visits 5 and 10 for withdrawal symptom ($r=0.597$, $P<0.05$) and HAD (anxiety) ($r=0.753$, $P<0.01$). Since the withdrawal symptom scale and the HAD scales were the only patient-rated scales used in the study, it appears that subjectively perceived anxiety was related to clinical course and outcome. Although there was no significant difference between placebo and buspirone groups in the ratings on these scales at the start of withdrawal (visit 5, Table II) the HAD (anxiety) was about 40% higher in the buspirone group before withdrawal (visit 0, Table II) and it is possible that such higher pre-withdrawal levels of subjective anxiety may have contributed to outcome in this group.

Plasma concentrations

Although initial diazepam plasma concentration was significantly higher in the buspirone group, there was no significant relationship between this concentration or that of nordiazepam and outcome variables, irrespective of treatment. Furthermore, there was no significant difference in the rate of fall of these plasma concentrations after diazepam withdrawal between groups: between visit 3 (starting level) and visit 9 (end of diazepam withdrawal) the mean fall in plasma diazepam concentration was 51.6%, and in nordiazepam 48.7% in the placebo group; corresponding figures in the buspirone group were 39.3% for diazepam and 43% for nordiazepam.

Characteristics of buspirone

No pharmacokinetic interaction between buspirone and diazepam was noted. Furthermore, no specific symptoms related to buspirone were observed. Although nausea, dizziness, headache, nervousness, insomnia, light-headedness and excitement have been described during buspirone treatment (Lader, 1988; *The Lancet*, 1988), scores on these separate items were not significantly different between placebo and buspirone groups. However, one patient, who withdrew after four weeks of buspirone treatment, reported headaches, periorbital numbness and dizziness which occurred regularly after taking each buspirone dose and lasted for about one hour. It is also possible that buspirone may have exacerbated withdrawal effects (see Discussion).

Psychiatric and personality factors

The diagnoses of the patients are listed in Table V. All patients fulfilled DSM–III criteria for hypnotic dependence (code number 304.11) at entry into the trial. The administration of SCID enables a diagnosis to be made before benzodiazepines were started. The difference in outcome of patients receiving placebo compared with those taking buspirone was not due to differences in the diagnoses of the patients allocated. Although four of the six patients with a diagnosis of agoraphobia with panic attacks dropped out of the trial, there were three patients with this diagnosis in each group at the start of the trial. Of the seven patients with personality disorder, five received placebo and all these completed the trial successfully.

TABLE V

DSM–III diagnosis before administration of benzodiazepines

Diagnosis	*DSM–III code*	*No. entering trial (n = 23)*	*No. completing trial (n = 15)*
Agoraphobia with panic attacks	300.21	6	2
Major depression, recurrent	296.32	6	4
Generalised anxiety disorder	300.02	5	4
Panic disorder	300.01	3	2
Dysthymic disorder	300.40	1	1
Adjustment disorder with anxious mood	309.24	1	1
Social phobia	300.23	1	1

There was no relationship between the presence or type of personality disorder and outcome. Of the eight patients who failed to complete the trial, four had normal personalities, two had personality difficulties, one had a schizoid personality disorder and one had a passive dependent disorder (personality difficulty refers to a tendency towards a personality type which is not sufficiently extreme to be outstanding to others). Six of the patients who completed the trial successfully had a personality disorder, including three with passive dependent personality disorder, of whom two had gross disorder as measured on the Personality Assessment Schedule. The patients with personality disorder had a trend towards higher initial anxiety ratings at the start of withdrawal compared with those with normal personalities (HAD anxiety, visit 5: personality disorder 13.9 (s.d. 7.3), normal personality 9.4 (s.d. 5.4)). Two of the patients who entered the trial had sociopathic personality difficulty; both these patients failed to complete. Patients with personality disorder showed a similar degree of dysphoria and withdrawal symptoms during the trial compared to those with normal personalities.

Discussion

This study shows that the course and outcome of benzodiazepine withdrawal was considerably worse in patients receiving buspirone before, during and after the withdrawal period than in those receiving placebo under double-blind conditions. Only four of the 11 patients taking buspirone completed the trial, compared with 11 of the 12 patients taking placebo, and in those continuing with the trial, withdrawal symptoms were more severe in the buspirone group. Only six of the 11 buspirone patients were off benzodiazepines at six and 12 months, compared with 11 of the 12 on placebo. These results are broadly similar to those of Olajide & Lader (1987*a*) who also found a higher failure rate in patients taking buspirone during benzodiazepine withdrawal compared with those taking placebo.

In the present study initial diazepam dosage and plasma concentration were significantly higher in the buspirone group. However, when all patients were considered irrespective of treatment, there was no significant relation between starting diazepam dose or plasma concentration and dropout rate or outcome at six and 12 months, and no significant correlation between pre-withdrawal diazepam dosage and withdrawal symptoms or psychiatric ratings. Hence the discrepancies in initial diazepam dosage and plasma concentrations could not account for the difference in outcome variables between the two groups.

At the start of benzodiazepine withdrawal the buspirone group had a (non-significant) trend towards higher anxiety scores on the HAD (anxiety) scale than the placebo group and there was a significant correlation overall between high HAD (anxiety) scores and poorer clinical outcome. Higher levels of subjectively perceived anxiety may have partially accounted for poorer outcome in the buspirone group, but it is possible that buspirone may also have exacerbated withdrawal effects. Buspirone has been noted to be less effective as an anxiolytic in patients who have previously taken benzodiazepines (Schweizer *et al*, 1986; Cohn *et al*, 1986; Lader, 1987) and has been reported to be dysphoric and anxiogenic in some patients (Bond & Lader, 1981). Buspirone is a mixed agonist/antagonist at 5-HT_{1A} receptor sites (Chopin & Briley, 1987; Fozard, 1987) and it is possible that under some circumstances, such as benzodiazepine withdrawal, antagonist rather than agonist effects predominate.

Acknowledgements

Our thanks are due to Dr H. W. Griffiths, Consultant Psychiatrist at St George's Hospital, Morpeth and Dr T. Packer, Consultant Psychiatrist, Royal Devon and Exeter Hospital, formerly Senior Registrars on the Northern Regional Psychiatric Rotating Scheme, for valuable assistance in the rating of patients. We thank Bristol Myers CNS for supplying the buspirone and placebo tablets and for laboratory and administration expenses.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Ashton, H. (1984) Benzodiazepine withdrawal: an unfinished story. *British Medical Journal*, **288**, 1135–1140.

Bond, A. J. & Lader, M. H. (1981) Comparative effects of diazepam and buspirone on subjective feelings, psychological tests and the EEG. *International Pharmacopsychiatry*, **16**, 212–220.

Chopin, P. & Briley, M. (1987) Animal models of anxiety: the effect of compounds that modify 5-HT transmission. *Trends in Pharmacological Sciences*, **8**, 383–390.

Cohn, J. B., Bowden, C. L., Fisher, J. G., *et al* (1986) Double-blind comparison of buspirone and clorazepate in anxious out-patients. *American Journal of Medicine*, **80**, 10–16.

Dourish, J. R., Hutson, P. H. & Curzon, G. (1986) Putative anxiolytics 8-OH-DPAT, buspirone and TVXQ 7821 are agonists at 5-HT_{1A} autoreceptors in the raphe nuclei. *Trends in Pharmacological Sciences*, 7, 212–214.

Eison, A. S. & Temple, D. L. (1986) Buspirone: review of its pharmacology and current perspectives in its mechanism of action. *American Journal of Medicine*, **80**, 1–9.

Feighner, J. P., Meridith, C. H. & Hendrickson, G. A. (1982) A double-blind comparison of buspirone and diazepam in outpatients with generalised anxiety disorder. *Journal of Clinical Psychiatry*, **43**, 103–107.

Fozard, J. R. (1987) 5HT: the enigma variations. *Trends in Pharmacological Sciences*, **8**, 501–506.

Lader, M. (1987) Long-term anxiolytic therapy: the issue of drug withdrawal. *Journal of Clinical Psychiatry*, **48**, 12–16.

—— (1988) The practical use of buspirone. In *Buspirone: A New Introduction to the Treatment of Anxiety* (ed. M. Lader), pp 71–76. London: Royal Society of Medicine.

Montgomery, S. & Asberg, M. (1979) A new depression scale designed to be sensitive to change. *British Journal of Psychiatry*, **134**, 382–389.

Murphy, S. M., Owen, R. & Tyrer, P. (1989) Comparative assessment of efficacy and withdrawal symptoms after six and twelve weeks treatment with diazepam or buspirone. *British Journal of Psychiatry*, **154**, 529–534.

Olajide, D. & Lader, M. (1987*a*) A comparative study of the efficacy of buspirone in relieving benzodiazepine withdrawal symptoms. *Journal of Clinical Psychopharmacology*, **7**, 11–15.

—— & —— (1987*b*) A comparison of buspirone, diazepam and placebo in patients with chronic anxiety states. *Journal of Clinical Psychopharmacology*, **7**, 148–152.

—— (1988) Buspirone as an alternative to the benzodiazepines: how effective? Abstracts of British Association for Psychopharmacology Summer Meeting, Jesus College, Cambridge, July 10–13. *Journal of Psychopharmacology*, **2**.

Raskin, A., Schulterbrandt, J., Reatig, N., *et al* (1969) Replication of factors of psychopathology in interview, ward behaviour and self-report ratings of hospitalised depressives. *Journal of Nervous Mental Disorders*, **148**, 87–89.

Rickels, K., Weisman, K., Norstad, N., *et al* (1982) Buspirone and diazepam in anxiety: a controlled study. *Journal of Clinical Psychiatry*, **43**, 81–86.

Schweizer, E., Rickels, K. & Lucki, J. (1986) Resistance to the anti-anxiety effect of buspirone in patients with a history of benzodiazepine use. *New England Journal of Medicine*, **314**, 717–720.

Spitzer, R. L., Williams, J. B. W. & Gibbon, M. (1985) *Instruction Manual for the Structured Clinical Interview for DSM-III-R (SCID, 7.1.85 revision)*. New York: Biometrics Research Department. New York State Psychiatric Institute.

TAYLOR, D. P., HYSLOP, D. K. & RIBLET, L. A. (1980) Buspirone: a new non-benzodiazepine anxiolytic. *Transactions of the American Society of Neurochemistry*, 11, 193.

THE LANCET (1988) Buspirone – a radical advance in the treatment of anxiety? *Lancet*, *i*, 801–806.

TRABER, J. & GLASER, T. (1987) 5-HT_{1A} receptor-related anxiolytics. *Trends in Pharmacological Sciences*, 8, 432–437.

TYRER, P. & ALEXANDER, J. (1979) Classification of personality disorder. *British Journal of Psychiatry*, 135, 168–174.

—— & OWEN, R. (1984) Anxiety in primary care: is short-term drug treatment appropriate? *Journal of Psychiatric Research*, 18, 73–78.

TYRER, P., OWEN, R. T. & CICCHETTI, D. V. (1984) The brief scale for anxiety: a subdivision of the comprehensive psychopathological rating scale. *Journal of Neurology, Neurosurgery, and Psychiatry*, 47, 970–975.

ZIGMOND, A. S. & SNAITH, R. P. (1983) The hospital anxiety and depression (HAD) scale. *Acta Psychiatrica Scandinavica*, 67, 361–370.

*C. H. Ashton, DM, FRCP, *Reader in Clinical Psychopharmacology, Clinical Psychopharmacology Unit, Department of Pharmacological Sciences, University of Newcastle upon Tyne, NE2 4HH*; M. D. Rawlins, BSc, MD, FRCP, *Professor of Clinical Pharmacology, University of Newcastle upon Tyne*; S. P. Tyrer, MB, LMCC, FRCPsych, *Consultant Psychiatrist, Royal Victoria Infirmary, Newcastle upon Tyne*

*Correspondence

British Journal of Psychiatry (1990), **157**, 239–248

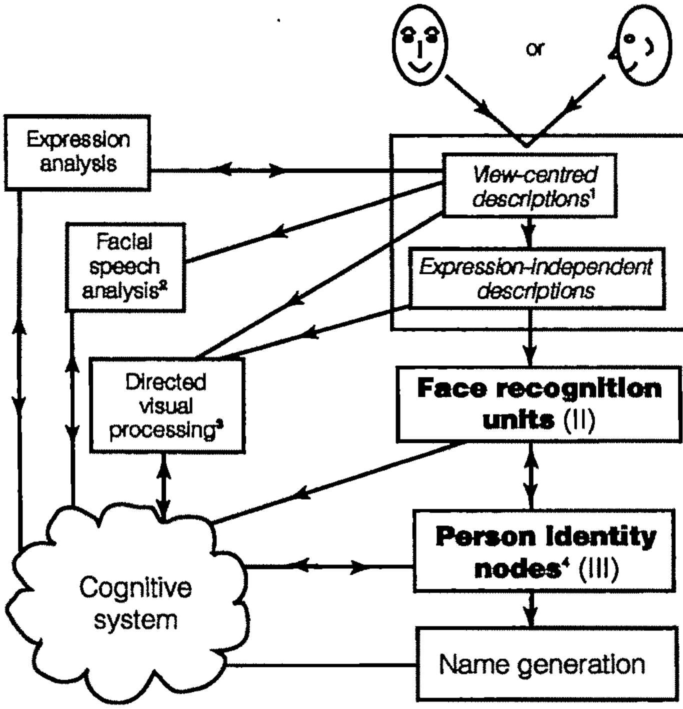
Accounting for Delusional Misidentifications

HADYN D. ELLIS and ANDREW W. YOUNG

Accounts of the major DMSs are given using theoretical models of the functional components underlying recognition of familiar people. Thus, Capgras' syndrome is suggested to involve impairment of processes that can support 'covert' recognition of familiar faces in prosopagnosia. It therefore forms a potential 'mirror image' of the impairments underlying prosopagnosia, and earlier attempts to link the two conditions directly are questioned. Frégoli syndrome and intermetamorphosis are explained as defects at different stages of an information-processing chain. Not only are these accounts consistent with the association of different DMSs with different brain injuries, but they also offer both suggestions for new inquiries and predictions about possible preserved and impaired abilities.

The delusional misidentification syndromes (DMSs) are psychiatric disorders distinguished by the fact that they all involve some deviation from normal processes of recognising people. A recent collection of papers on DMS (Christodoulou, 1986) gives a good coverage of the history, findings and issues, and Young & Ellis (1989*a*) discuss theoretical explanations for the way people process physiognomic information. As yet, however, the theoretical insights gained from psychological and neurological studies have had little impact on the traditionally psychiatric field of DMS.

Before discussing three of the most prominent DMS disorders (Capgras', Frégoli and intermetamorphosis), we first describe the model of face recognition by Bruce & Young (1986), which extends that by Hay & Young (1982) and is the best summary of available data. More extensive discussions of such models are available elsewhere (e.g. Bruce, 1988; Young & Ellis, 1989*a*; Ellis, 1989). Here, we simply focus on those


or
Expression analysis
Facial speech analysis²
Directed visual processing³
View-centred descriptions¹
Expression-independent descriptions
Structural encoding (I)
Face recognition units (II)
Person identity nodes⁴ (III)
Name generation
Cognitive system

1. These represent the object as it is actually seen
2. Analysis of facial movement aspects of speech
3. Analysis for age, sex, race, etc.
4. These can also be accessed by voice, gait, etc. (Ellis, 1989) independent of the first two stages

FIG. 1. The information-processing model of familiar face recognition (Bruce & Young, 1986).

parts of the model that are relevant for our attempts to provide theoretical explanations for DMS. Following this, we consider the neurological condition of prosopagnosia in order both to underline the conclusions of the face-recognition model and to provide the specific hypothesis for our analysis of Capgras' syndrome.

A model of face recognition

It is possible to attempt a theoretical explanation of DMSs within the kind of information-processing model of face recognition put forward by Bruce & Young (1986*a*). According to this model (Fig. 1) the recognition of a familiar face involves sequential stages. Firstly, there is a stage (I) in which the information concerning the structure of the seen face is encoded using 'descriptions' which are view-centred (i.e. represent the object as it is actually seen) and/or independent of expression. These descriptions provide raw data which are then analysed for expression, facial speech and information about age, sex, race, etc. Marr's (1982) model of vision was adapted for this stage. The second stage (II) is where the familiarity or otherwise of a face is signalled by the Face Recognition Units (FRUs). Familiar faces then stimulate information held at the Person Identity Node (PIN) level (III), where semantic or biographical information is stored. This third stage may also be accessed by voice or gait or other means of signalling identity. According to Hay & Young (1982), Bruce & Young (1986*a*), and Ellis (1986*a*), other aspects of face processing, such as the perception of facial expression, are accomplished independently from and in parallel with the determination of individual identity. Furthermore, information about the person's name is stored and retrieved independent of his or her biographical details.

Support for the Bruce & Young (1986*a*) model of face recognition comes from a diary study of everyday errors of identification (Young *et al*, 1985). This revealed problems including diarists recognising a face as being familiar but being unable to access biographical information concerning the person, and, more often, them knowing not only that the face is familiar but also all sorts of biographical details, yet not being able to locate the individual's name. Such experiences are consistent with the idea that the recognition process can become 'blocked' at each of the successive stages shown in Fig. 1.

Other experiments using reaction-time techniques have also produced data consistent with a sequential series of decision stages each of which takes a finite time to complete in a prescribed order (see Bruce, 1988; Sergent, 1986). The existence of independent modules for analysing other aspects of faces than identity (categorical definitions of age, sex, race, etc. and facial expression) are also supported by latency data (see Bruce, 1986; Young *et al*, 1986; Bruce *et al*, 1988).

Much of the strongest evidence for this model however, comes from neurological case histories of patients suffering from prosopagnosia, the inability to recognise previously familiar faces (Bodamer, 1947) which usually follows bilateral occipital-inferotemporal damage (Meadows, 1974). This condition produces a more or less complete breakdown of overt face recognition ability in which even the most familiar faces – friends, relatives, family, and the patient's own face when seen in a mirror – are not consciously recognised. (For a history of the disorder, see Ellis, 1989.) Typically, prosopagnosic patients fall into one of two or more groups depending on whether their symptoms are primarily perceptual, whereby they see faces as blobs, caricatures, all alike, etc., or more memory based, whereby faces look normal enough but evoke no sense of familiarity (Hécaen, 1981; De Renzi, 1986).

The majority of prosopagnosics have suffered strokes and are middle-aged to elderly men (Mazzuchi & Biber, 1985). Usually, bilateral areas of occipito-temporal cortex are involved (Meadows, 1974; Damasio *et al*, 1982), but in some cases neuroimaging techniques have revealed occipito-temporal lesions involving only the right cerebral hemisphere (De Renzi, 1986; Landis *et al*, 1986). Most prosopagnosic patients manage to identify familiar people on the basis of voice, gait and clothing which suggests that the damage occurs before the PIN stage of processing. Some display problems in identifying facial expression (Shuttleworth *et al*, 1982) but most prosopagnosics can interpret the moods of others based upon physiognomic cues (Bruyer *et al*, 1983). Face recognition and expression interpretation, however, are doubly dissociable, implying separate mechanisms (Kurucz & Feldmar, 1979). By the same token, face recognition and the ability to make use of lip-reading to aid normal communication also show double dissociation (Campbell *et al*, 1986). As Ellis (1986*b*) has argued, these different cases lend support to the various stages of the model shown in Fig. 1.

Theoretical approach to understanding DMS

Our aim in this paper is to advance explanations for the major DMSs based on the current understanding of normal face-recognition processes and the ways in which they can be impaired. In doing this, our explicit intention is to extend the cognitive neuropsychological approach to the study of conditions that have been traditionally defined as being psychiatric in nature.

Recent developments in cognitive neuropsychology have leant heavily on the view that complex mental functions involve the interaction of a number of modular subcomponents (Fodor, 1983; Ellis & Young, 1988; Shallice, 1988). The neurological and neurophysiological literature now provides abundant evidence for such modular organisation, which would make sense in terms of the important 'design principles' of keeping essential neural interconnections as short as possible and allowing part of a complex system to be modified by experience or evolution without adversely affecting the performance of the rest of the system (Marr, 1982; Cowey, 1985).

Studies of the different types of breakdown that can affect performance of any complex cognitive capacity can thus provide insight into the underlying organisation of the responsible functional components. More importantly, an adequate theoretical model should be able to account for the types of impairment that can occur and in turn, should itself be modified if inconsistent forms of impairment are actually observed (Ellis & Young, 1988).

Here we try to demonstrate that this approach which has been successful in understanding the breakdown of normal performance, both in terms of everyday slips and errors and the deleterious effects of brain injury, can also be of benefit in understanding delusional misidentifications. In doing so, we do not entirely take a leap into the dark, since much recent evidence has begun to suggest some organic involvement in DMS, and parallels to neurological conditions have already been drawn. Furthermore, such an approach, carried out systematically, has advantages in allowing a more theoretical understanding of the conditions, in suggesting aspects worthy of further investigation and in predicting certain patterns of co-occurring symptoms.

The last point leads us to an important caveat. It is traditional to refer to delusional misidentification *syndromes*, but in fact, unlike when they were first introduced, each of these is now actually defined by a single particular symptom, rather than a symptom constellation. We would thus prefer to speak of Capgras *symptom*, and so on, but have retained the conventional 'syndrome' terminology here because it has become so thoroughly entrenched in the literature.

The major DMSs

Capgras' syndrome

In 1923 Capgras & Reboul-Lachaux reported the case of a 53-year-old woman who displayed what they called "l'illusion des sosies", a delusional belief that people who she knew had been replaced by identical doubles (see Berson, 1983, for an explanation of 'sosies' and Christodoulou, 1986, for the historical/mythological background). In addition to believing that her husband and children had been replaced by doubles, the woman later claimed that police and neighbours had been similarly duplicated. She also believed that there existed doubles of herself.

Berson (1983) reports a computer search which located 133 similar case reports in English, as well as reports in French, Italian, Russian, Dutch, German, Spanish and Japanese; and Signer (1987) found reports of 315 patients in the English and French literature. In most cases the diagnosis was of schizophrenia. Many cases also appeared to involve organic disorders, but, regardless of the cause of Capgras' syndrome, the symptoms of most patients are dominated by a marked paranoid component (Berson, 1983). It is also worth noting that the people believed to have been duplicated are generally those close to the patients. These are the kind of people for whom the patient has strongly positive or negative affective responses. Sometimes patients believe that there is more than a single impersonator (Todd *et al*, 1981).

Capgras' patients tend to be paranoid, or, at least, rather suspicious by nature, and the doubles are invariably thought of as evil or dangerous in some way. Capgras' patients also tend to suffer from depersonalisation–derealisation. Patients sometimes report that everything looks strange, for example, things may look painted or not natural and faces may look like masks or wax models or seem to have been changed by plastic surgery. Interestingly, the instance of Capgras' among females is generally thought to be rather higher than it is among males (Sims & White, 1973).

Organic v. functional aetiology of Capgras' syndrome

Although labelling Capgras' syndrome as a manifestation of paranoid schizophrenia has constituted an explanation of the disorder for some commentators, it is a rather unsatisfactory and incomplete one. Merrin & Silberfarb (1976), whose ideas were more impressive, saw a connection between depersonalisation–derealisation and Capgras' syndrome symptoms when they wrote "a number of cases began with reduced feelings of unreality or depersonalisation, (were) followed by indiscriminate misidentification and finally by the establishment of the Capgras' delusion". However, as Todd *et al* (1981) point out, this in itself does not fully explain the development of Capgras' syndrome, because patients afflicted by feelings of depersonalisation or derealisation have insight and appreciate the illusory nature of the phenomenon, whereas those with Capgras' delusion generally don't.

In the main, Capgras' syndrome has been treated as a functional disorder. A number of investigators have noted, however, that Capgras' patients often have associated brain lesions, or that their symptoms may have resulted from some form of poisoning or other exogenous factor. In his review of focal abnormalities of the central nervous system in patients with a DMS, Joseph (1986) discovered that the majority of patients have clear computerised tomography (CT) scan evidence of brain abnormalities. Of the 23 patients studied by him, approximately two-thirds had signs of cortical atrophy. In all cases the signs were bilateral: in some they were largely frontal; in others parietal or temporal. Unfortunately, Joseph did not break down his group by specific DMS so there is no way of attributing specific sites of brain damage to particular types of DMS. It may be noted that Kiriakos & Aranth (1980) found obvious evidence of organic damage in only four out of 13 Capgras' patients, but they employed X-ray and electroencephalography (EEG) measures – rather gross forms of imaging – whereas many lesions require CT scan or magnetic resonance imaging (MRI) to become evident. Joseph (personal communication, 1989) claims that all DMS patients given full evaluation have revealed some CNS abnormality.

Fishbain *et al* (1986) reported the case of someone displaying Capgras' syndrome following metrizanide myelography. The patient, a 67-year-old male, had been admitted to hospital following pain in his left calf. Twenty hours after myelography, the patient developed the following symptoms: severe vomiting, severe headache, confusion, disorientation to time and place (he thought he was in his son's home) and a delusional belief that his doctor was a duplicate of the original. After treatment with prochlorperazine, symptoms gradually remitted.

MacCallum (1973) also reported Capgras' syndrome following medical intervention; in this case it appeared to be due to the patient inhaling an overdose of a bronchial dilator containing adrenaline and adropinemethonitrate. In another of MacCallum's cases, Capgras' syndrome appeared to be related to the patient's diabetes. A patient described by Hay *et al* (1974) showed Capgras-like symptoms following electroconvulsive therapy (ECT). All of these instances cast doubt on Enoch's (1963) suggestion that the title 'Capgras' syndrome' should be reserved for the delusional misidentification and replacement for an identical double in a clear sensorium, and this *without organic basis* (i.e. in a setting of functional illness with a psychodynamic interpretation).

Explaining Capgras' syndrome

As we have indicated earlier, a number of researchers now take the view that Capgras' syndrome may be the result of underlying organic brain damage. Although the evidence for this is not yet conclusive, modern brain-imaging techniques have shown that in a large number of cases of Capgras' syndrome there is clear evidence of brain lesions. Nonetheless, some commentators, such as Bersen (1983), argue that "organic factors in themselves, however, seem neither necessary nor sufficient to explain the particular and peculiar content of the delusion". This view is perhaps overstated.

One of the most interesting suggestions put forward by those who favour an organic basis for Capgras' syndrome is that the condition is related to one long recognised in the neurological literature, known as reduplicative paramnesia (Alexander *et al*, 1979). Such paramnesias often involve reduplication of places, with the patient maintaining that he/she is in a place which is an almost exact copy of the actual location. There is a clear parallel with the Capgras' delusion, which involves reduplication of people, and Alexander *et al* (1979) demonstrated that a combination of bilateral frontal and right hemisphere damage seemed to be present for patients of both types. The link between the two conditions is supported by Anderson (1988), who drew attention to a number of cases in which Capgras' patients were observed to reduplicate more than just people. Interestingly, Kapur *et al* (1988) reported the case of a 71-year-old man who developed reduplicative paramnesia for places but not faces following right frontal vascular lesion, indicating the dissociable nature of reduplicative paramnesia.

A link between prosopagnosia and Capgras' syndrome has also been suggested, but never successfully forged. Lewis (1987) in his study of a single case of transient Capgras' syndrome, for example, provided MRI data demonstrating the presence of bilateral occipito-temporal lesions as well as smaller bilateral frontal lesions. The frontal lesions are consistent with the findings of Alexander *et al* (1979) but occipito-temporal lesions are the usual anatomical correlate of prosopagnosia (Meadows, 1974; Damasio *et al*, 1982). As we have said, the occipito-temporal lesions underlying prosopagnosia are often bilateral, but in some cases (based only on evidence from brain imaging) are apparently located exclusively in the right cerebral hemisphere (De Renzi, 1986; Landis *et al*, 1986).

The patient studied by Lewis (1987) had actually shown signs of mild prosopagnosia during childhood, and this prompted him to suggest some sort of connection between the two syndromes. According to Lewis there seems to be a parallel between prosopagnosia and Capgras' syndrome. In the latter case there would appear to be a disconnection between the percept of a face and its evocation of affective memories.

Joseph (1986) also posited a disconnection explanation. "It may be that in misidentification syndromes in particular and reduplicative phenomena in general, brain disease causes a disconnection between the right and left hemisphere cortical areas, that decode afferent sensory information and maintain the normal functions of orientation to person, place, time and object relationship that we describe as 'orientation'." In Joseph's view each hemisphere forms a representation of a face separately. Normally, these images are 'fused' by interhemispheric transfer, and thus are presented on a conscious level as a fully integrated representation of the external world. "In patients with misidentification syndromes . . . each hemispheric 'image' is presented separately leading to an awareness of the conscious level of two simultaneously separate but physically identifiable or similar persons, places, objects, times or object relationships. Depending upon which hemispheric connections are most impaired, the clinical syndromes of misidentification, reduplication, or disorientation will result". Although there is as yet no very compelling empirical evidence to support Joseph's view, it merits further investigation and is not necessarily inconsistent with the theoretical ideas which we present shortly.

Face-processing impairments in Capgras' patients

There seems little doubt that face-processing impairments can be found in Capgras' patients. Tzavaras *et al* (1986) found that on face-matching tests (Tzavaras *et al*, 1970) DMS patients were significantly poorer than pathological and normal controls. They deemed this "an infra-clinical prosopagnosic symptom" which may imply some organic factor in DMS. Shraberg & Weitzel (1979) studied two Capgras' patients by giving them the Benton face-matching test (Benton *et al*, 1983) which they claimed was particularly sensitive to prosopagnosia. Finding that their Capgras' patients also performed poorly on this test they then argued that a parallel existed between the two syndromes. However, as Benton (1980) has emphasised, the face-matching test is not sensitive to prosopagnosia: many, if not most, prosopagnosics can adequately perform the test (Bauer, 1984; Young & Ellis, 1989*b*), although they may rely on unusual strategies (Newcombe, 1979). Instead, it is patients with right parietal lesions who perform such tasks especially poorly; yet these are not prosopagnosic (i.e. they remain able to recognise familiar faces). In the same way, it should be emphasised that patients who experience the Capgras' delusion are not prosopagnosic: they continue to recognise without apparent difficulty the dummies and imposters that have replaced their relatives. So if there is any connection between DMS and prosopagnosia it is unlikely to be a direct one.

Other misconceptions of the nature of prosopagnosia have also been introduced into the literature on Capgras' syndrome. For example, Todd *et al* (1981) maintain that in order for an organic aetiology for Capgras' syndrome to be established, it is necessary to show a clear cause and effect relationship between prosopagnosia and Capgras' syndrome. They even add that the association is particularly doubtful in view of the fact that there are usually no signs of any visual field defects in Capgras' patients. Here they appear to believe, erroneously, that visual field defects are a necessary concomitant of prosopagnosia. This is not the case. Prosopagnosia can exist in the absence of visual field defects, although they frequently accompany the condition but not, it would seem, in any causally connected way (Meadows, 1974).

If there is a connection between prosopagnosia and Capgras' syndrome, then it operates in a rather more subtle way. In order to develop this argument, it is necessary to consider evidence for different recognition routes, derived from the phenomena of 'covert recognition' of faces by prosopagnosics. Bauer (1984) studied a prosopagnosic patient, LF, who had suffered bilateral occipito-temporal brain damage following a motorcycle accident. He was profoundly prosopagnosic, and yet when shown pictures of previously familiar faces (famous or personally known), along with five names drawn from the same semantic category, which were read out to him, LF displayed significantly greater autonomic responses to the correct name compared with the four incorrect names. This technique, known as the Guilty Knowledge Test, is widely used by American police forces as a method for detecting lying by criminal suspects. Bauer's data mainly concern the skin conductance response (SCR). LF showed a discriminating SCR to some 62.5% of the known faces – well above any chance level of performance (i.e. 20%). Tranel & Damasio (1985) confirmed Bauer's observations using a modified technique in which a series of randomly arranged known and unknown faces were presented to two prosopagnosic patients. They also showed increased autonomic reactions to the previously known faces.

Bauer (1984, 1986) advanced the view that there are two routes to facial recognition. The main route runs from visual cortex to temporal lobes via the inferior longitudinal fasciculus. Following Bear (1983), he termed this the 'ventral route': it corresponds to the system responsible for overt or conscious recognition, and it is the route which typically is damaged in cases of prosopagnosia. The other, described as the 'dorsal route', runs between the visual cortex and the limbic system, via the inferior parietal lobule, and is sometimes intact in prosopagnosic patients. It is this latter route which, Bauer claims, gives the face its emotional significance and hence, when the ventral route is selectively damaged, can give rise to covert recognition (i.e. recognition at an unconscious level).

If we accept Bauer's dual recognition route (and from the work of De Haan *et al*, 1987, it may be that we should be looking for more than two routes), we can try to apply the notion towards an understanding of Capgras' syndrome. Young & Ellis (1989*a*) have begun to explore the phenomenon of Capgras' syndrome by suggesting that it is a mirror image of prosopagnosia. In other words, patients with Capgras' syndrome seem to have an intact primary or ventral route to face recognition, but may have a disconnection along or damage within the secondary or dorsal route. This would mean that they receive a veridical image of the person they are looking at, which stimulates all the appropriate overt semantic data held about that person, but they lack another, possibly confirming, set of information which, as Lewis (1987) and Bauer (1986) have independently suggested, may carry some sort of affective tone. When patients find themselves in such a conflict (that is, receiving some information which indicates the face in front of them belongs to X, but not receiving confirmation of this), they may adopt some sort of rationalisation strategy in which the individual before them is deemed to be an imposter, a dummy, a robot, or whatever extant technology may suggest.

Derombies (1935) also attached great importance to the affective state of Capgras' patients. He suggested that Capgras' syndrome results from a simultaneous intellectual recognition and affectively engendered non-recognition of faces: at one level the patient recognises the face but at another level he or she does not recognise it. More recently (and also independently) Anderson (1988) made a similar suggestion when he argued "that the Capgras' delusion results from lesions of the pathway for visual recognition at a stage where visual images are imbued with affective familiarity". Anderson's ideas (and those of Derombies) are similar to the ones presented above although he (1988) is not able to be more specific about the mechanism by which "affective familiarity" may operate. Indeed, one difference is that our hypothesis rests on the view that more than one recognition pathway is involved, and makes the clear prediction that Capgras' patients will *not* show the normally appropriate skin conductance responses to familiar faces, despite the fact that these will be overtly recognised.

The explanation, however, is still incomplete. Bersen (1983) pointed out that in Capgras' syndrome, not all faces seem to be duplicated, only those of people close to the patient. A possible answer to this is that it is only in these instances that one expects more than the basic information about a face. Only certain faces normally have associated with them a particularly strong affective component, and therefore only these are vulnerable in the case of any disconnection between the visual areas and the cerebral structures involved in Bauer's dorsal recognition pathway.

One may extend this argument to other reduplicative paramnesias. Places, objects, etc. are not affectively neutral and so the absence of an emotionally charged input could produce the feeling of recognition, but it not being quite right. Again this would be particularly true for those places and objects with which the patient is most familiar.

Frégoli syndrome

In 1927 Courbon & Fail reported another form of DMS. Their patient was a 27-year-old labourer's daughter who had worked variously as a domestic servant, in a factory, in a restaurant, etc. She was paid by the day and slept in Salvation Army type refuges. Her abiding passion was the theatre; she preferred to go without food in order to attend a performance. Admitted to hospital following an attack on an employer, she reported to Courbon & Fail that she was the victim of enemies: in particular of the actresses Robine and Sarah Bernhardt whom she had often seen in the theatre. She insisted that these actresses had followed her about for many years, taking the form of people she knew or met, overpowering her thoughts, making her do this instead of that and stroking her or forcing her to masturbate. The patient reported often being interfered with or attacked by people who particularly took the form of Robine.

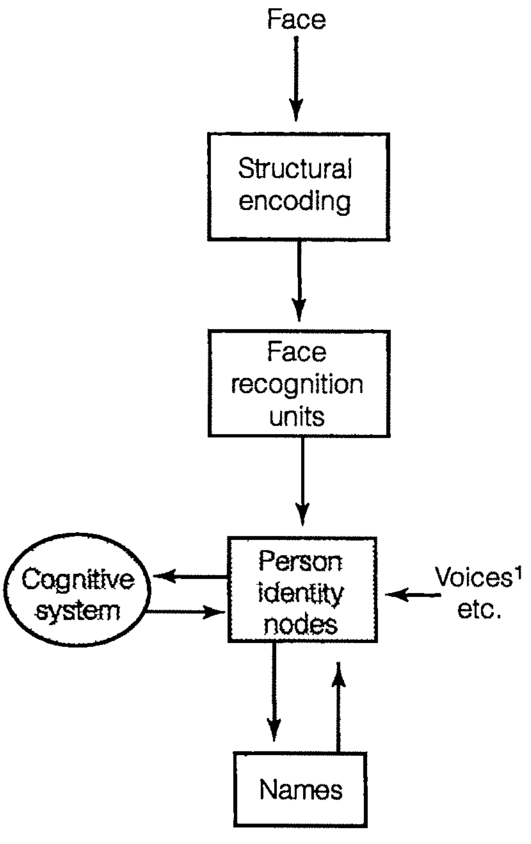
Courbon & Fail adopted the term 'Frégoli syndrome' after the Italian actor and mimic, Leopoldo Frégoli, famous for his ability to impersonate people on the stage. The hallmark of the Frégoli syndrome is the delusional misidentification of familiar persons disguised as others. De Pauw *et al* (1987) classified the illusion of Frégoli as hyper-identification. They contrast this with Capgras' syndrome, which was classified as hypo-identification. They reported the case of Mrs C, a 66-year-old widow, who believed that she was being persecuted by her cousin and his female friend. She described in detail how the couple disguised themselves with make-up, wigs, dark glasses, false beards and different clothes, and repeatedly accosted her when she was out. She confronted strangers in public demanding that they reveal their true identity; she took complicated routes on her way home to shake off her persecutors; and she often reported their activities to the local police. "They keep changing their clothes and their hairstyles, but I know it's them. He can look like an old man. They want a medal for doing that. It's like an actor and actress preparing for different scenes," she said.

Mrs C had a history of brain damage: she suffered haematoma over the left frontal area after a fall in 1982. A CT scan also showed right-sided posterior-temporo-parietal infarct together with general cortical atrophy. She had earlier been treated for hypertension and she had also been diagnosed as having right-sided temporal arteritis. Although she recovered from her initial symptoms of speech slurring and memory problems as well as her orientation difficulties, she then developed the delusional belief just described. Her Frégoli syndrome seemed to be controllable with trifluoperazine.

As in cases of Capgras' syndrome, Frégoli may be associated with some degree of objective face-processing impairment. De Pauw *et al* (1987) found that Mrs C scored poorly on the Warrington face-recognition test. More detailed investigation of such impairments would be worthwhile.

Frégoli syndrome bears some relation to a type of error that all of us make in everyday life (Young *et al*, 1985). When we expect to meet a particular person, occasionally and transiently we will misidentify a stranger as that person. However, we usually quickly correct this mistake when inconsistent evidence is picked up. In the Frégoli syndrome, on the other hand, such inconsistencies are also noted, but attributed to the effects of disguise. The syndrome can thus be characterised as involving impaired decision mechanisms. In Bruce & Young's terms, the impairment is in the cognitive system itself, which places an inappropriate evaluation on the evidence it receives. Such decision mechanisms can also malfunction for normal people, as other everyday errors collected by Young *et al* (1985) showed.

The PIN stage and associated cognitive system would therefore be the likely site for malfunction leading to Frégoli syndrome (see Fig. 2). Specifically, either the PIN system becomes driven by a deranged cognitive system or hyperexcitable nodes representing particular people appear to become engaged, sometimes almost regardless of whatever output from the previous FRU stage occurs, and impaired decision mechanisms accept this evidence. Since strangers' faces



1. Not shown in original model.

Fig. 2. Simplified face-recognition model indicating the level at which it is proposed the illusion of Frégoli occurs.

are often the source of the patients' delusions it is clear that it would be of interest to study whether any degree of resemblance to the misidentified person is necessary.

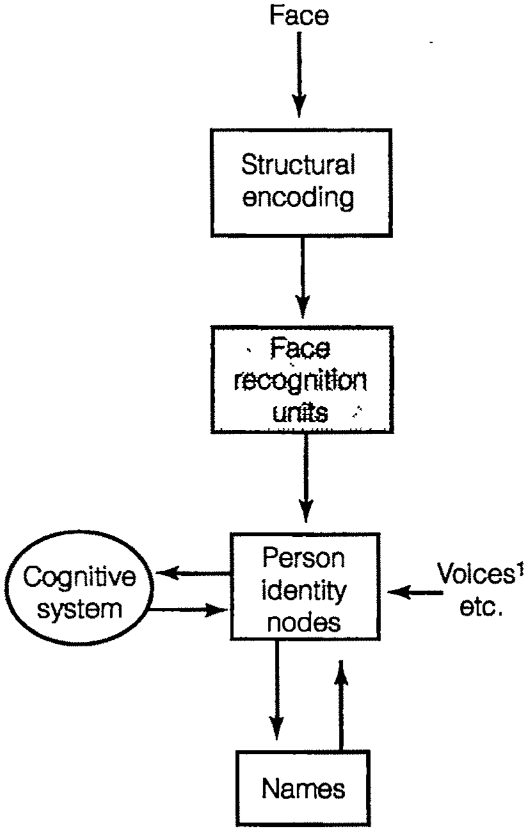
Intermetamorphosis

First described by Courbon & Tusques (1932), intermetamorphosis refers to cases where, for the patient, the physical appearance of some people may change radically to correspond with the appearance of someone else. Courbon & Tusques described a 49-year-old depressive patient who reported that animals and objects she owned took the form of another animal or object. She also experienced changes in her husband's appearance, which could become exactly like that of a neighbour or could rapidly transform to look larger or smaller or younger. Various people in rapid succession also took on the guise of her son, but she used the particularly large size of his feet to discount these delusions.

Courbon & Tusques (1932) pointed out that intermetamorphosis involves a false recognition of both appearance and associated identity, whereas in the illusion of Frégoli the patient is not confused about physical appearance. Intermetamorphosis is relatively rare: Bick (1986) reviewed two cases reported after Courbon & Tusques' (Christodoulou, 1975; Malliaros *et al*, 1978) and presented another, a 42-year-old woman who had had a long history of epilepsy and paranoid schizophrenia. During a period in hospital, she became suddenly agitated and declared that her doctor was her dead uncle.

According to Bick (1986), three of the four cases reviewed by him had temporal lobe epilepsy. Joseph (1987) has subsequently described a 49-year-old patient who displayed both Capgras' syndrome (believing her husband had been replaced by an identical imposter) and intermetamorphosis (stating that several other patients looked like her father and that her son's physical appearance had altered). In this case a CT scan was given and it revealed mild atrophy of the frontal lobe, severe bilateral atrophy of the temporal lobe, moderate atrophy of the occipital lobe and a mildly enlarged ventricular system. The *prima facie* evidence for a possible organic component in intermetamorphosis, as for other DMS disorders, is thus quite strong.

In order to establish an explanation for intermetamorphosis within the Bruce & Young (1986) model of normal face recognition processes, it is necessary to focus on the second stage, the FRU stage, highlighted in Fig. 3. Representations for known faces are posited to exist, each of which is somehow excited or triggered by the appearance of the particular person. The FRUs then signal familiarity and, in turn, communicate with appropriate PINs in the semantic memory system where the person's biographical details are stored.



1. Not shown in original model.

FIG. 3. Simplified face recognition model indicating the level at which it is proposed intermetamorphosis occurs.

Intermetamorphosis may be construed as an inappropriate excitation of an FRU which could occur, for example, if the particular FRU had a considerably lowered triggering threshold. A corollary to this suggestion is that it is more likely that faces similar to the misidentified person will cause the unit to fire inappropriately, and, as with the Frégoli symptom, more systematic investigation of the resemblance between the appearance of the actual and delusionally substituted people would be useful. A clear prediction from our account of these conditions is that physical resemblance will be important in triggering episodes of intermetamorphosis but much less important to the Frégoli delusion.

At this stage it is not possible to establish why and how such threshold shifts occur, but notice that once an FRU has fired it will automatically then cause the corresponding and equally inappropriate PIN to become excited. Unfortunately, none of the case histories of patients' behaviour during such an instance of misidentification has reported how the patients responded to voices. According to the model shown in Fig. 3, voices input from a different recognition system but employ the same PINs. Joseph & O'Leary (1987) indeed reported a Frégoli case who claimed that a Mr B had been replaced by another acquaintance. While the physical appearance of Mr B was unchanged he appeared to have the other person's voice. Thus, whereas Frégoli patients may misperceive voices, those with intermetamorphosis syndrome could make an incorrect facial identification but should not produce the same error on the voice. This should produce discrepancies at times which would, perhaps, be resolved because of the primacy of visual over aural perception but could, nonetheless, be elicited during clinical interview.

Conclusion

The advantages in attempting to explain various DMS states within the theoretical constructs derived from studies of normal and pathological face recognition are numerous. Chief among them, of course, is the hope that it may lead both to a complete understanding of DMSs and, in turn, assist clinicians in diagnosis and treatment.

Secondly, the opportunity to bring together a seemingly disparate set of phenomena, for some time effectively dealt with as being largely unrelated, carries with it many advantages. For example, the ideas that have been posited regarding Capgras' syndrome allow us to perceive a very obvious deficit in the model outlined in Fig. 1, that is, it only depicts the 'overt' route to face recognition. Experiments on prosopagnosic patients, together with the theoretical ideas we have presented here, strongly suggest that the model is incomplete in that it only displays a single route to recognition and fails to accommodate what, perhaps, may turn out to be a sort of emotional recognition of faces (and indeed other routes, as well).

The third aspect of our endeavours to which we wish to draw attention is its potential value in organising clinical observations of DMS patients. We have suggested various types of evidence that should be collected routinely in such cases. It is, for instance, clearly important to note patients' responses to voices as well as faces, because by doing so we may refine our ideas concerning the level of impairment. Some assessment of the similarity between people mistakenly perceived in intermetamorphosis and wrongly categorised in Frégoli syndrome would also be of theoretical benefit.

The final point we wish to stress is that discussing DMS in relation to prosopagnosia reinforces the suggestions made by Joseph (1986) and others that DMS may originate primarily from organic bases. The next step must surely be to derive routinely in cases of DMS high-quality brain images that should assist in classification. In time it should be possible to correlate particular DMSs with damage to specific brain areas.

Acknowledgements

The authors are funded by an Economic and Social Research Council Programme: Recognising Faces (XC15250004). They would like to thank Drs Szulecka and De Pauw for their help and encouragement.

References

Alexander, M. P., Stuss, D. T. & Benson, D. F (1979) Capgras syndrome: a reduplicative phenomenon. *Neurology*, 29, 334–339

Anderson, D. N. (1988) The delusion of inanimate doubles: implications for understanding the Capgras phenomenon. *British Journal of Psychiatry*, 153, 694–699.

Bauer, R. M. (1984) Autonomic recognition of names and faces: a neuropsychological application of the Guilty Knowledge Test. *Neuropsychologia*, 22, 457–469.

—— (1986) The cognitive psychophysiology of prosopagnosia. In *Aspects of Face Processing* (eds H. Ellis, M. Jeeves, F. Newcombe, *et al*). Dordrecht: Martinus Nijhoff.

Bear, D. M. (1983) Hemispheric specialization and the neurology of emotion. *Archives of Neurology*, 40, 195–202.

Benton, A. L. (1980) The neuropsychology of facial recognition. *American Psychologist*, 35, 176–186.

——, Hamsher, K., Varney, N. R., *et al* (1983) *Contributions to Neurological Assessment*. New York: Oxford University Press.

Berson, R. J. (1983) Capgras' syndrome. *American Journal of Psychiatry*, 140, 8.

Bick, P. A. (1986) The syndrome of intermetamorphosis. *Bibliotheca Psychiatrica*, 164, 131–135.

Bodamer, J. (1947) Die Prosop-Agnosie. *Archiv fur Psychiatrie und Nervenkrankheiten*, 179, 6–54.

Bruce, V. (1986) Recognising familiar faces. In *Aspects of Face Processing* (eds H. Ellis, M. Jeeves, F. Newcombe, *et al*). Dordrecht: Martinus Nijhoff.

—— (1988) *Recognising Faces*. Hillsdale, NJ: Lawrence Erlbaum.

—— & Young, A. (1986) Understanding face recognition. *British Journal of Psychology*, 77, 305–327.

——, Ellis, H., Gibling, F., *et al* (1988) Parallel processing of the sex and familiarity of faces. *Canadian Journal of Psychology*, 41, 510–520.

Bruyer, R., Laterre, C., Seron, X., *et al* (1983) A case of prosopagnosia with some preserved covert remembrance of familiar faces. *Brain and Cognition*, 2, 257–284.

Campbell, R., Landis, T. & Regard, M. (1986) Face recognition and lip reading: a neurological dissociation. *Brain*, 109, 509–521

Capgras, J. & Reboul-Lachaux, J. (1923) L'illusion des 'sosies' dans un délire systématise chronique. *Bulletin de la Société Clinique de Médecine Mentale*, 2, 6–16.

Christodoulou, G. W. (1978) Syndrome of subjective doubles. *American Journal of Psychiatry*, 135, 249–251.

Christodoulou, G. N. (1975) Reported in Bick (1986).

—— (1986) The delusional misidentification syndrome. *Bibliotheca Psychiatrica*, **164**.

Courbon, P. & Fail, G. (1927) Illusion of Frégoli syndrome and schizophrenia. *Societe Clinique de Médecine Mentale.*

—— & Tusques, J. (1932) Illusions d'intermetamorphose et de charme. *Annales Medico-Psychologiques*, **14**, 401–406.

Cowey, A. (1985) Aspects of cortical organization related to selective attention and selective impairments of visual perception: A tutorial review. In *Attention and Performance, XI* (eds M. Posner & O. Marin). New Jersey: Erlbaum.

Damasio, A. R., Damasio, H & Van Hoesen, G. W. (1982) Prosopagnosia: anatomical basis and behavioral mechanisms. *Neurology*, **32**, 331–341.

De Haan, E., Young, A. & Newcombe, F. (1987) Face recognition without awareness *Cognitive Neuropsychology*, **4**, 385–415.

De Pauw, K. W., Szulecka, T K. & Poltock, T. L. (1987) Single case study. Frégoli syndrome after cerebral infarction. *Journal of Nervous and Mental Disease*, **175**, 1–6.

De Renzi, E. (1986) Current issues in prosopagnosia. In *Aspects of Face Processing* (eds H. Ellis, M. Jeeves, F. Newcombe, *et al*). Dordrecht: Martinus Nijhoff.

Derombies, M. (1935) L'illusion de sosie, forme particuliers de la reconnaissance systematique (these de Paris quoted by A. Brochado). *Annales Medico-Psychologiques*, *94*, 706–717).

Ellis, A. W. & Young, A. G. (1988) *Human Cognitive Neuropsychology*. London: Erlbaum.

Ellis, H. D. (1986*a*) Processes underlying face recognition. In *The Neuropsychology of Face Perception and Facial Expression* (ed. R. Bruyer). Hillsdale, NJ: Lawrence Erlbaum.

—— (1986*b*) Disorders of face recognition. In *Neurology* (eds K. Poek, H. J. Freund & H. Ganshirt). Heidelberg: Springer-Verlag.

—— (1989) Past and recent studies of prosopagnosia. In *Developments in Clinical and Experimental Neuropsychology* (eds J. R. Crawford & D. M. Parker). New York: Plenum.

Enoch, M. (1963) The Capgras syndrome. *Acta Psychiatrica Scandinavica*, **39**, 437–462.

Fishbain, D. A. & Rosomoff, H. (1986) Capgras syndrome associated with metrizamide myelography. *International Journal of Psychiatry in Medicine*, **16**, 131–136.

Fodor, J (1983) *The Modularity of Mind*. Cambridge, Mass: MIT Press.

Hay, D. C. & Young, A. W. (1982) The human face. In *Normality and Pathology in Cognitive Functions* (ed. A. Ellis). New York: Academic Press.

Hay, G. G., Jolley, D. J. & Jones, R. G. (1974) A case of Capgras' syndrome in association with pseudo-hypoparathyroidism. *Acta Psychiatrica Scandinavica*, **50**, 73–77.

Hécaen, H. (1981) The neuropsychology of face recognition. In *Perceiving and Remembering Faces* (eds G. Davies, H. Ellis & J. Shepherd). London: Academic Press.

Joseph, A. B (1986) Focal central nervous system abnormalities in patients with misidentification syndromes. *Bibliotheca Psychiatrica*, **164**, 68–79.

—— (1987) Delusional misidentification of the Capgras and intermetamorphosis types responding to clorazepate. *Acta Psychiatrica Scandinavica*, **75**, 330–332.

—— & O'Leary, D. H. (1987) Anterior cortical atrophy in Frégoli syndrome. *Journal of Clinical Psychiatry*, **48**, 409–411.

Kapur, N , Turner, A & King, C. (1988) Reduplicative paramnesia: possible anatomical and neuropsychological mechanisms. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 579–581.

Kiriakos, R. & Ananth, J. (1980) Review of 13 cases of Capgras syndrome. *American Journal of Psychiatry*, **137**, 1605–1607.

Kurucz, J. & Feldmar, G. (1979) Proso-affective agnosia as a symptom of cerebral organic disease. *Journal of the American Geriatrics Society*, **27**, 225–230.

Landis, T., Cummings, J. G., Christen, L., *et al* (1986) Are unilateral right posterior cerebral lesions sufficient to cause prosopagnosia? Clinical and radiological findings in six additional patients. *Cortex*, **22**, 243–252.

Lewis, S. W. (1987) Brain imaging in a case of Capgras' syndrome. *British Journal of Psychiatry*, **150**, 117–121.

MacCallum, W. A. G. (1973) Capgras symptoms with an organic basis. *British Journal of Psychiatry*, **123**, 639–642.

Malliaras, D. E., Kossovitsa, Y. T. & Christodoulou, G N. (1978) Organic contributors to the intermetamorphosis syndrome. *American Journal of Psychiatry*, **135**, 8.

Marr, D. (1982) *Vision*. San Francisco: Freeman.

Mazzuchi, A. & Biber, C. (1983) Is prosopagnosia more frequent in males than females? *Cortex*, **19**, 509–516.

Meadows, J. C. (1974) The anatomical basis of prosopagnosia. *Journal of Neurology, Neurosurgery and Psychiatry*, **37**, 489–501.

Merrin, E. L. & Silberfarb, P. M. (1976) The Capgras phenomenon. *Archives of General Psychiatry*, **33**, 965–968.

Newcombe, F. (1979) The processing of visual information in prosopagnosia and acquired dyslexia: Functional versus physiological interpretation. In *Research in Psychology and Medicine I* (eds D. Osborne, M. Gruneberg & J. Eiser). London. Academic Press

Sergent, J. (1986) Methodological constraints on neuropsychological studies of face perception in normals. In *The Neuropsychology of Face Perception and Facial Expression* (ed R. Bruyer). Hillsdale, NJ: Erlbaum.

Shallice, T. (1988) *From Neuropsychology to Mental Structure.* Cambridge: Cambridge University Press.

Shraberg, D. & Weitzel, W. D. (1979) Prosopagnosia and the Capgras syndrome. *Journal of Clinical Psychiatry*, **40**, 313–316.

Shuttleworth, E. C., Syring, V. & Allen, N. (1982) Further observations on the nature of prosopagnosia. *Brain and Cognition*, **1**, 307–322.

Signer, S. F. (1987) Capgras syndrome: the delusion of substitution.*Journal of Clinical Psychiatry*, **48**, 4.

Sims, A. & White. (1973) Coexistence of the Capgras and de Clerambault syndromes. A case history. *British Journal of Psychiatry*, **123**, 635–637.

Todd, J., Dewhurst, K. & Wallis, D. (1981) The syndrome of Capgras. *British Journal of Psychiatry*, **139**, 319–327.

Tranel, D. & Damasio, A. R. (1985) Knowledge without awareness: an autonomic index of facial recognition by prosopagnosics. *Science*, **228**, 1453–1454.

Tzavaras, A., Hécaen, H. & LeBras, H. (1970) Le problème de la specificité du déficit de la reconnaissance du visage humain lors des lésions hemisphériques unilatérales *Neuropsychologia*, **8**, 403–418.

——, Luate, J. & Bidault, E. (1986) Face recognition dysfunction and delusional misidentification syndromes. In *Aspects of Face Processing* (eds H. Ellis, M. Jeeves, F. Newcombe, *et al*). Dordrecht: Martinus Nijhoff.

Young, A. W. & Ellis, H. D. (1989*a*) *Handbook of Research on Face Processing*. Amsterdam: North Holland.

—— & —— (1989*b*) Childhood prosopagnosia. *Brain and Cognition*, **9**, 16–47.

——, Hay, D. C. & Ellis, A. W. (1985) The faces that launched a thousand slips: everyday difficulties and errors in recognizing people. *British Journal of Psychology*, **76**, 495–523.

Young, A. W., McWeeny, Hay, D. C., *et al* (1986) Matching familiar and unfamiliar faces on identity and expression. *Psychological Research*, **48**, 63–68.

*Hadyn D. Ellis, BA, PhD, DSc, CPsychol, FBPsS, *School of Psychology, University of Wales College of Cardiff, Cardiff CF1 3YG*; Andrew W. Young, BSc, PhD, DSc, CPsychol, FBPsS, *Department of Psychology University of Durham, Durham DHE 3LJ*

*Correspondence

British Journal of Psychiatry (1990), 157, 249–260

Factors Related to Self-reporting of the Pre-menstrual Syndrome

PAMELA WARNER and JOHN BANCROFT

Menstrual health questionnaires were completed by a self-selected sample of the readership of a woman's magazine ($n = 5457$). Sixty-one per cent of subjects described themselves as suffering from pre-menstrual syndrome (PMS) and this was largely corroborated by ratings of symptoms pre-menstrually, menstrually and post-menstrually for the most recent cycle. Mood symptoms were more strongly implicated than physical ones. Self-report of PMS was found to be modestly associated with aspects of parity and oral contraceptive use, but strongly and positively related to the duration of 'natural' menstrual cycles (i.e. uninterrupted by pregnancy or steroidal contraception) and to psychosocial stress. There were interactions among psychosocial factors and between psychosocial load and duration of natural cycles.

The concept of 'pre-menstrual syndrome' (PMS) remains unclear. Definitions vary, and it is questionable whether PMS is a discrete entity or covers a variety of cyclical phenomena with possibly different aetiologies. This has profound implications for research, in particular for 'measurement' and 'confirmation' of PMS. Even in treatment studies on 'confirmed-PMS' subjects the response to a variety of treatment methods is unpredictable, and the literature on controlled-outcome studies is confused, with disagreement probably reflecting crucial differences in patient characteristics which are obscured by the global concept of PMS (Bancroft & Backstrom, 1985).

Homogeneity of symptoms does not necessarily imply homogeneity of aetiology, and differences in aetiology could account for some of the variations in response to different treatment regimes. Conversely, a particular aetiological mechanism may be expressed as a number of different symptom patterns depending on interactions with personality and health. Over 150 symptoms have been implicated in PMS, and each sufferer will have her own particular diagnostic 'justification'. Schilling (1981) found strong support for cycle-related variability in most subjects, but with considerable between-subject differences in the nature of the reaction pattern. Some of the reported failure to confirm self-designation as a PMS sufferer by rating symptoms may result from lack of sensitivity of the 'summary' criteria applied. Hamilton & Alagna (1988) have argued forcefully that while highly selected 'confirmed-PMS' samples are appropriate for biomedically-orientated treatment studies, to seek such a sample is inappropriate and indeed premature in research aimed at exploring the nature and variability of symptoms. Thus, although the more typical symptom patterns of PMS are well known, there is no theoretical basis for designating them the *sine qua non*. Indeed, to do so when exploring aetiological mechanisms might well pre-empt the potential findings.

PMS has been found to be associated with a number of factors such as age, parity, psychopathology, menstrual characteristics, employment and variants of stress (Logue & Moos, 1986; Bancroft & Backstrom, 1985). These factors are themselves interrelated and so a large-scale survey would be required to explore aetiological links. Retrospective self-report is the only measurement method practicable for large surveys. While this method, applied to menstrual-cycle phenomena, has been criticised, it is generally believed to be fairly reliable for women with moderate to severe symptoms, particularly if it pertains to a woman's 'last' cycle rather than some 'usual' experience over a number of recent cycles (see Endicott & Halbreich, 1982; Logue & Moos, 1986). Two studies which have utilised rigorous analysis (of equivalent measures) have somewhat restored the reputation of retrospective reporting. Schilling (1981) found consistency between daily and retrospective reports, and little evidence for stereotypic distortion of such reports. Hart *et al* (1987) found that recall of a cycle accounted for 72% of the variance in that cycle's prospective scores, whereas prospective scores from one cycle predicted only 14% of the variance in scores for the next cycle. In addition, many subjects who make daily ratings of symptoms for one or more cycles experience a reduction in their pre-menstrual changes (Endicott & Halbreich, 1988). Thus, low inter-cycle reliability in the syndrome and the therapeutic effect of prospective record-keeping may each account for some reports of failure prospectively to confirm retrospective ratings of PMS. Nevertheless, potential sources of distortion in the use of retrospective ratings should be kept in mind.

In the study reported here, a large sample spanning a wide age range has been obtained through the readership of a women's magazine. Such a self-selected sample, with largely unknown selection biases, will not be representative of either the readership or of women in general. Clearly a population probability sample would be preferable, but these are very expensive on a large scale and would be difficult to justify until we know more about the aetiology and 'measurement' of PMS. While our 'readership' sample does make possible multifactorial exploratory analyses, the results reported should be regarded as principally hypothesis generating, and significant findings will require replication. In this paper we report the relationship between the self-designated status of a 'PMS sufferer', the types and timing of specific symptoms reported, and variables such as age, parity, contraceptive method and employment status.

Method

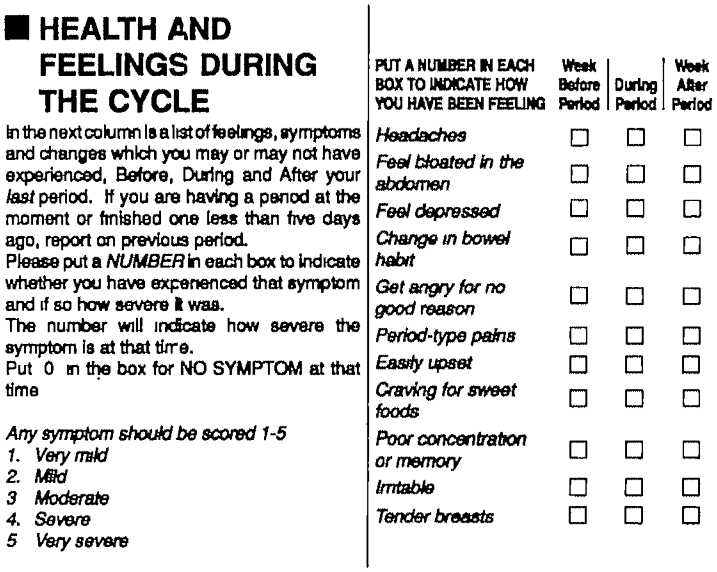
The questionnaire

In August 1985 a detailed menstrual health questionnaire was published in a national women's weekly magazine (*Woman*, UK). The accompanying feature on PMS included some general information, but deliberately avoided being specific about symptoms or their timing and duration. Readers were invited to help medical research by completing the questionnaire. It was laid out in the style normally utilised by the magazine for reader surveys, with the questions grouped into sections. In order, these were: 'About you' covering current marital status/relationship, parity and periods; 'Health and feelings during the cycle' which comprised a list of 27 symptoms to be rated for three time periods, the week before, the time during and the week after the last menstrual period; 'Pre-menstrual syndrome' which asked whether the subject believed she suffered PMS, and if so, further questions about her experience of it; and 'General questions' covering employment, age, contraceptive method and its duration of use. The method of symptom rating in the second section is new and was developed for this survey in a pilot study of 250 women attending a family planning clinic. Part of this section is shown in Fig. 1, and all but the four least prevalent symptoms (allergic reactions, infections, craving for salt and for 'other' foods) are listed in Table I.

Subjects and analysis

The average weekly sales of the magazine at the time was about two-and-a-half million. The questionnaire was long and detailed. Factors influencing the decision to complete it would include literacy and an interest in menstrual-cycle changes, with a majority having personal experience of such changes. A total of 7620 completed questionnaires were returned, a record response for any comparable survey conducted by the magazine.

A sample of 997 forms, representative of the readership, was taken by the magazine for its own report and follow-up feature. Of the remainder, 360 were unusable because of inadequate or incorrect completion, or because the woman was pregnant or had had a hysterectomy. A further 806 forms were excluded from this analysis on account of self-report of 'a great deal of (unusual) stress at the present time' or because their relationship with their partner was rated 'not at all happy'. Such severe life stresses are at the very least likely to confound symptom reporting, but are also implicated in the exacerbation of PMS (Woods *et al*,



■ HEALTH AND FEELINGS DURING THE CYCLE

In the next column is a list of feelings, symptoms and changes which you may or may not have experienced, Before, During and After your *last* period. If you are having a period at the moment or finished one less than five days ago, report on previous period.
Please put a *NUMBER* in each box to indicate whether you have experienced that symptom and if so how severe it was.
The number will indicate how severe the symptom is at that time.
Put 0 in the box for NO SYMPTOM at that time

Any symptom should be scored 1-5

1. *Very mild*
2. *Mild*
3. *Moderate*
4. *Severe*
5. *Very severe*

PUT A NUMBER IN EACH BOX TO INDICATE HOW YOU HAVE BEEN FEELING	Week Before Period	During Period	Week After Period
Headaches	☐	☐	☐
Feel bloated in the abdomen	☐	☐	☐
Feel depressed	☐	☐	☐
Change in bowel habit	☐	☐	☐
Get angry for no good reason	☐	☐	☐
Period-type pains	☐	☐	☐
Easily upset	☐	☐	☐
Craving for sweet foods	☐	☐	☐
Poor concentration or memory	☐	☐	☐
Irritable	☐	☐	☐
Tender breasts	☐	☐	☐

FIG. 1. Detail from published questionnaire showing method of symptom reporting.

1982) and the onset of affective disorder (Miller *et al*, 1989). Furthermore, a proportion of women attending PMS clinics are diagnosed as affectively ill (Endicott & Halbreich, 1988). Since a particular focus of this survey was the patterns of symptoms reported in PMS and the possibility of detecting subtypes of the syndrome (to be described in a further paper), it was decided that subjects who reported these severe life stresses should be excluded.

Analyses were therefore based on the remaining 5457 questionnaires, although for comparison, the age distribution and self-designated PMS status of the excluded groups were also reported. The data were analysed in the form of contingency tables with independence determined by the χ^2 test.

The multifactorial nature of the data dictates the use of controlling variables. Where a variation in association occurs across the layers of a controlling variable, χ^2 is presented separately for the different layers. Where layers have small n-values the tests will be of low power and, conversely, in tables with very large n-values, even modest associations can yield highly statistically significant results.

Results

Reported PMS-status

Each respondent was allocated to one of three groups on the basis of her answer to the question 'Do you believe you suffer from PMS now?'. Overall, 62% of all 7620 respondents answered 'yes', 31% 'uncertain/don't know' and 7% 'no'. Two of the excluded subgroups (the magazine sample and subjects ($n=302$) submitting incomplete/erroneous data) were almost identical with regard to PMS status (61% and 62% answered 'yes' respectively). Pregnant women ($n=53$) were less likely to answer 'yes' to PMS suffering *now* (51%). However, those subjects excluded on account of a relationship rated 'not at all happy' ($n=145$) or because they indicated they were currently undergoing 'a lot' of unusual stress ($n=661$) were significantly more likely to designate themselves as suffering from PMS (74% 'yes', $\chi^2=8.7$, 2 d.f., $P\leq0.025$; 70% 'yes', $\chi^2=21$, 2 d.f., $P<0.000$ respectively).

For the 5457 subjects included in the analysis, the self-assessed PMS-status was 61% 'yes', 32% 'uncertain/don't

TABLE I

Proportions of subjects with classification of symptoms as pre-menstrual, menstrual or post-menstrual (n = 5457)

Symptoms	*Proportion of subjects with 'severe' symptoms*				
	Pre-menstrual[1] %	*(Pre-menstrual*[2] *only)* %	*Menstrual*[3] %	*Post-menstrual*[4] %	*Total*[1+3+4] %
Mood symptoms					
Irritable	65	(43)	3	3	71
Get angry for no good reason	64	(47)	2	3	69
Easily upset	60	(41)	3	3	66
Feeling tense	52	(35)	3	3	58
Mood swings	52	(34)	2	4	58
Feel depressed	51	(39)	3	3	57
Feel bad about myself	46	(28)	3	4	53
Violent feelings	33	(24)	1	2	36
Other symptoms					
Feel bloated in the abdomen	45	(33)	5	1	51
Tender breasts	40	(29)	1	1	42
Craving for sweet foods	35	(25)	2	2	39
Poor concentration	31	(22)	3	2	36
Clumsiness	29	(20)	2	1	32
Passing water frequently	23	(13)	7	3	33
Backache	21	(9)	8	1	30
Headaches	19	(15)	5	3	27
General aches and pains	18	(10)	4	1	23
Period-type pains	18	(9)	23	1	42
Spots e.g. acne	16	(10)	1	2	19
Difficulty in sleeping	15	(11)	2	2	19
Hot flushes/cold sweats	14	(8)	3	1	18
Change in bowel habit	13	(9)	6	1	20
Nausea/sickness	10	(6)	4	1	15

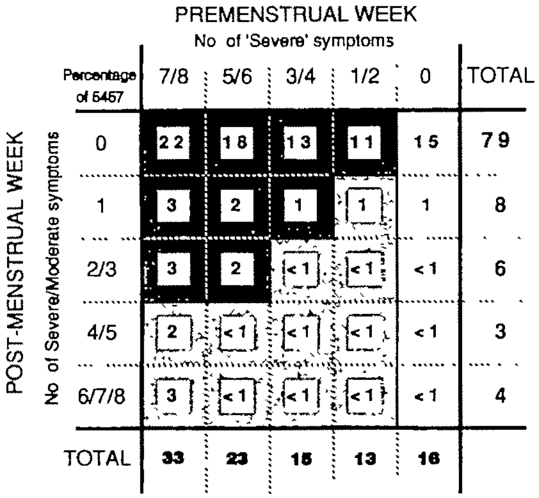
1. 'Severe' pre-menstrually, may continue during menstruation but not post-menstrually.
2. 'Severe' pre-menstrually but neither menstrually nor post-menstrually.
3. 'Severe' only during menstruation.
4. Occurs or persists 'severely' post-menstrually.

know', and 7% 'no'. Women who had described themselves as having PMS were asked to give their reason. Overall, 19% of them believed their PMS had been diagnosed by a doctor, and for a further 69% the basis was either "the changes are so regular it must be" or "I have been checking on myself".

Reported symptoms

Table I lists 23 symptoms from the questionnaire. Each subject's ratings for the three phases have been classified separately for each symptom as: 'not severe', if none of the three ratings was severe or very severe; 'menstrual' if severe only during menstruation; 'post-menstrual' if the symptom either persists or occurs severely after menstruation; or 'pre-menstrual', severe the week before but not the week following menstruation (with no account being taken of the severity of the symptom *during* menses). The proportions of subjects in each classification other than 'not severe' are given in Table I. The 'pre-menstrual' classification can be further subdivided according to whether or not the symptom persists severely during menstruation, the latter also given in Table I.

Mood symptoms were most commonly reported, in particular irritability and anger (65% and 64% 'pre-menstrual' respectively). There were eight symptoms related to mood, and the number of these rated severe pre-menstrually ranged from none (16% of respondents) to seven or eight (32%). The distribution was somewhat J-shaped, with 'no symptoms' being slightly more likely than one or two (the actual percentages can be ascertained from Fig. 2). There was a strong relationship between self-assessed PMS-status and the number of mood symptoms reported 'severe' pre-menstrually (Spearman $r = 0.37$, $P = 0.0000$).



PREMENSTRUAL WEEK
No of 'Severe' symptoms

POST-MENSTRUAL WEEK
No of Severe/Moderate symptoms

Percentage of 5457	7/8	5/6	3/4	1/2	0	TOTAL
0	22	18	13	11	15	79
1	3	2	1	1	1	8
2/3	3	2	<1	<1	<1	6
4/5	2	<1	<1	<1	<1	3
6/7/8	3	<1	<1	<1	<1	4
TOTAL	33	23	15	13	16	

FIG. 2. Cross-tabulation of all subjects by number of severe mood symptoms reported pre-menstrually against the number of moderate to severe symptoms post-menstrually (cells and marginal totals are expressed as % of $n = 5457$) symptom reports are further categorized as 'pre-menstrual pattern' (■) 75%; 'chronic tendency', where the symptoms do not remit sufficiently post-menstrually (▨), 16%; and 'no (severe) pre-menstrual symptoms' (□) 9%.

The most prevalent physical symptoms were abdominal bloating and breast tenderness (45% and 40% 'pre-menstrual' respectively). Considering the four most prevalent non-mood symptoms, together with period-type pains, subjects were classified according to the number of these five symptoms rated severe pre-menstrually but not post-menstrually: no symptoms, 25%; one symptom, 24%; two, 23%; and three or more, 28%. Thus these physical symptoms were more likely than the mood symptoms to occur independently of each other. Nevertheless, there was significant association between the number of these 'physical' symptoms and PMS-status (Spearman $r = 0.27$, $P = 0.0000$), although PMS-status was less strongly associated with physical symptoms than mood symptoms.

Since women had been asked specifically to rate symptoms for their most recent cycle they were also given the opportunity to note whether the symptoms reported were the same 'as usual'. Overall 13% felt this cycle had been 'better' and 16% 'worse' than usual.

Association of reported PMS status and symptoms

PMS is broadly defined as emotional and/or physical symptoms occurring pre-menstrually but largely remitting at some other phase of the cycle, usually during the follicular phase (which approximates to the week after menstruation). For the eight mood symptoms in our questionnaire, Fig. 2 shows the cross-classification of all subjects by the numbers of *severe* symptoms reported pre-menstrually against the number of *moderate or severe* symptoms post-menstrually (expressed as a percentage of 5457). As can be seen, 79% of subjects reported themselves as free of any mood symptoms, even to a moderate degree, post-menstrually. Superimposed on this cross-classification is an *ad hoc* grouping of subjects on the basis of their global mood symptom reporting, as follows: 'no (severe) pre-menstrual symptoms' (16%); 'pre-menstrual' pattern of mood symptoms (75%); and 'chronic tendency', where there is insufficient remission of symptoms post-menstrually (9%).

This symptom grouping largely corroborates the subjects' self-assessed PMS-status. Nevertheless, of the women who believed they did suffer PMS ('yes'), 17% did not report a 'pre-menstrual' pattern of mood symptoms (approximately half of these fall into the 'chronic tendency' subgroup and the remainder into the 'no (pre-menstrual) mood symptoms' subgroup). If severe pre-menstrual ratings for at least one physical symptom (of the five detailed above) is considered to be evidence of PMS in the absence of a pre-menstrual pattern of mood symptoms, then the proportion uncorroborated reduces to 6%. A further 2% said their most recent cycle was 'better' than usual whereas 1% appeared to be confusing PMS with dysmenorrhoea (i.e. predominantly menstrual phase symptoms including severe period pain). After allowing for these, there remains 3% of respondents whose self-description as suffering from PMS cannot be adequately explained on the basis of the 13 symptoms considered. In the 'PMS-maybe' group the corresponding proportion unexplained was 7%.

Conversely, 26% of the 359 women who did *not* assess themselves as PMS sufferers nevertheless reported a pre-menstrual pattern of mood symptoms. An additional 4% recorded two or more severe pre-menstrual physical symptoms, giving a total of 110 (30%) 'false negatives'. Of these, 10% ($n = 11$) had judged the cycle rated as 'worse than usual'. Among the remainder there may have been some lack of knowledge about PMS or it may not have been felt that the pre-menstrual changes were severe enough to warrant categorisation as suffering from PMS. Certainly the women with 'pre-menstrual' mood pattern but answering 'no' to PMS were reporting fewer symptoms (Fig. 3: $\chi^2 = 326$, 6 d.f., $P = 0.0000$). Thus, based on their retrospective reporting of the timing and severity of specific symptoms, nearly all the women who described themselves as PMS sufferers met conventional criteria for PMS. The high proportion of 'false negatives' is attributable to subjects with fewer severe symptoms.

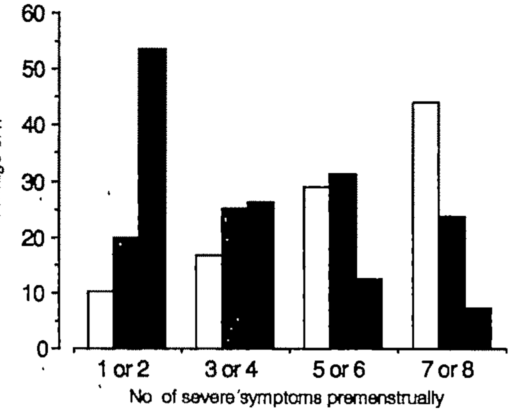


FIG. 3. Association between self-assessed PMS status and number of severe mood symptoms pre-menstrually in subjects with a 'pre-menstrual' pattern of mood symptoms ($\chi^2 = 326$, 6 d.f., $P = 0.0000$) (PMS-status: 'yes', $n = 2763$ (□); 'maybe', $n = 1229$ (▨); 'no', $n = 95$ (■)).

Age, contraception, parity and 'natural' cycles

Associations between these variables

As would be expected, contraceptive method is related to both age and parity (Table II). In particular, oral contraceptive (OC) use was far less common among parous than nulliparous women, and in both groups there was a sharp decline after 30 years of age. The differing distributions of age for parous and nulliparous women meant that age subgroups had to be constructed separately in each parity group. The number of subjects in each of these subgroups, and the broad age distribution in the two parity groups, are given in the last two columns of Table II. Overall, 4% of subjects were aged under 20 years and 13% 40 years and over. (There was a very similar age distribution to that shown in Table II in the groups excluded from the analysis on account of erroneous data, 'a great deal of stress', or for the magazine sample. The small group excluded on the grounds of 'unhappy relationship' comprised fewer young women.)

The age distribution of subjects included in the analysis was compared to the age distribution of the magazine readership, as estimated by market research, and the differences found were consistent with there having been a higher 'response rate' in the age band 25 to 35 years: this age band comprised 37% of the readership under 40 years of age compared to 54% of the respondents ($\chi^2 = 801$, 4 d.f., $P < 0.000$: comparison confined to those under 40 years of age to avoid effect of menopause on response rates in older women).

Of possible importance is the length of time that women have experienced uninterrupted 'natural' cycles (i.e. not pregnant or using hormonal contraception). For women not using hormonal contraception the current interval of 'natural' cycles was therefore

TABLE II
Contraception used by age and parity of subjects

Age/parity subgroups	*Contraceptive method (% of n)*							
	None	*Oral contraceptive*	*IUCD*[1]	*Female sterilisation*	*Vasectomy*	*Other*	n	*% of total ns*
Nulliparous								
Age: years								
≤24	32	45	2	0	1	20	894	43
25–29	33	29	6	1	2	29	643	31
≥30	48	13	5	4	6	24	550	26
						total n =	2087	
Parous								
Age: years								
≤29	15	22	19	7	13	24	724	21
30–34	10	8	14	16	26	26	1012	30
≥35	9	2	9	25	33	22	1634	49
						total n =	3370	

---

[1] Intra-uterine contraceptive device.

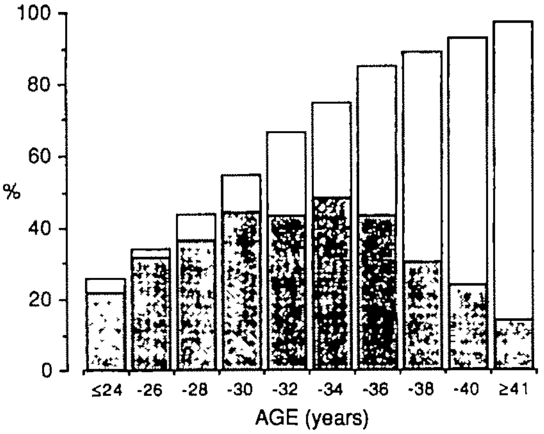


FIG. 4. Proportions (%) of parous women (not using oral contraceptives) with duration of natural cycles exceeding two years by age ($\chi^2 = 1145$, 18 d.f., $P = 0.0000$, $n = 3085$) (duration: 3–6 years (▨); >6 years (□)).

defined as the time since menarche, last childbirth or cessation of oral contraception, whichever was the most recent. This variable was strongly related to age (parous: $\chi^2 = 1145$, 18 d.f., $P = 0.0000$; nulliparous: $\chi^2 = 197$, 16 d.f., $P = 0.0000$). While it would be expected that the proportion who had experienced more than two years of natural cycles would increase steadily with age, it was striking that the proportion with three to six years of such cycles was maximal around 30 years of age (from 29 to 36 years of age for parous women, 27 to 34 years of age for nulliparous). This is illustrated for parous women in Fig. 4.

Associations with reported PMS status

Age. Table III gives the proportions of women reporting themselves to be PMS sufferers ('PMS-yes') for the parity pill subgroups subdivided by age. In all subgroups there was increased reporting with age, although in the parous-pill-using subgroup there were very few women in age groups other than 25–29 years and the χ^2 result is not statistically significant. In each subgroup the greatest increase occurred between the age groups on either side of 30 years of age. Almost half of all women under 20 years of age were 'uncertain' whether they suffered PMS. In both the parous and nulliparous groups 'uncertainty' diminished with age.

Natural cycles. For nulliparous women who were ex-pill users there was an association between PMS status and years since stopping oral contraception (overall $\chi^2 = 12.6$, 2 d.f., $P = 0.0018$, $n = 912$) such that the prevalence of PMS increased with duration of natural cycles. In order to ascertain more fairly the impact of this variable on perception of PMS, we repeated the analysis excluding women for whom duration of cycles was irrelevant (i.e. those women who had already assessed themselves as suffering from PMS before this interval of natural cycles). In the remaining women (without pre-existing PMS) the effect was even more striking and persisted when age was controlled. The overall effect for all ages combined is illustrated in Fig. 5*a* ($\chi^2 = 59.4$, 4 d.f. $P = 0.0000$, $n = 536$).

Among nulliparous women who had never used hormonal contraception, the duration of natural cycles dated from menarche, and given the narrow age range for this event, the effects of age and cycles were confounded. The prevalence of PMS ranged from 34% in the first six years of menstruation to 75% among women over 30 years who have the longest duration of cycles ($\chi^2 = 47.2$, 6 d.f., $P = 0.0000$, $n = 515$).

For parous women there is also an association between reporting of PMS and duration of natural cycles ($\chi^2 = 24.8$, 4 d.f., $P = 0.0001$, $n = 3085$). Here too, the effect is much more marked when the analysis is confined to women whose PMS was not pre-existing at the time of resumption of natural cycles. This was seen in each age group separately, and is illustrated in Fig. 5*b* for parous women aged 30–39 years ($\chi^2 = 68.6$, 4 d.f., $P = 0.0000$, $n = 1226$). Parous women could be subdivided according

TABLE III

Proportions of women (%) reporting 'PMS-yes' by age, parity and oral contraceptive use

Age group: years	*Proportion of women reporting 'PMS-yes'*							
	Nulliparous				*Parous*			
	Pill-users		*Non-pill-users*		*Pill-users*		*Non-pill-users*	
	% *of* n	(n)	% *of* n	(n)	% *of* n	(n)	% *of* n	(n)
⩽19	27	(73)	41***	(123)	48	(159)	58*	(565)
20–24	40	(325)	46	(373)				
25–29	46	(185)	56***	(458)				
30–34	56	(69)	70**	(481)	64	(84)	69*	(928)
35–39					50	(30)	71*	(989)
⩾40					—	(0)	70	(615)
PMS × Age								
χ^2 (6 d.f.)	13.3		67.5		10.6		37.9	
P	0.038		0.0000		0.099		0.0000	

Pill × non-pill χ^2 (2 d.f.): *$P < 0.06$; **$P < 0.01$; ***$P < 0.0005$.

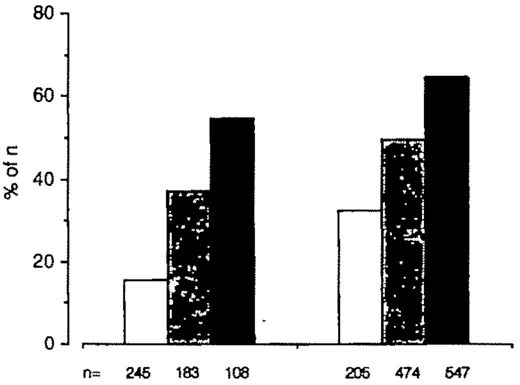


FIG. 5. Proportion (%) of women reporting 'PMS-yes' by duration of natural cycles in women without prior PMS (duration: 2 years (□); 3–6 years (▨); ≥7 years (■)).
(a) Nulliparous women, ex-oral-contraceptive users, all ages.
(b) Parous women aged 30–39 years.

to whether the last interruption to menstruation had been pregnancy (62%) or OC use since last birth (38%). In the former subgroup the age of youngest child is the same as the calculated duration of cycles whereas in the latter it is greater, by an amount depending on the timing and duration of that interval of OC use. The pattern of prevalence of PMS increasing with duration of natural cycles was similar in both subgroups, although in the post-pregnancy subgroup there was overall a higher prevalence of PMS (controlling for age). This was most noticeable in the early years of natural cycles (≤six years of cycles: 50% v. 35% 'PMS-yes', $\chi^2=10.8$, 2 d.f., $P=0.005$, $n=679$).

Hormonal contraception. Table III also shows that OC users are less likely to report PMS, the effect being much more marked in nulliparous women. However, in the younger nulliparous groups the most frequent reporting of PMS was among ex-pill users. (Current users 37%, never users 39%, ex-users 50%, $n=398$, 264, 232 respectively; $\chi^2=21$, 4 d.f., $P=0.0003$). This effect is all the more striking considering that ex-pill users will have experienced a shorter duration of natural cycles than never-users. In addition, 41% of those current pill-users who believed they were suffering from PMS dated its onset during their current period of OC use. The types of pill in current use were predominantly low oestrogen (≤30 mg), 48% of nulliparous and 35% of parous pill-users, and triphasic preparations, 36% and 33% respectively. High-oestrogen and progestagen-only pills were more commonly used by parous women and there was an association between pill-type and age: progestagen only and high oestrogen pill-users were more likely to be over 30 years of age and nulliparous triphasic pill users were more likely to be under 25 years of age. Although no firm conclusions are possible because of this and other confounding factors, there was no detectable association between pill type and self-report of PMS.

Parity. When age and OC use were controlled for, there was only a weak trend for parous women to be more likely to report PMS (Table III). The prevalence of PMS by number of children is given in Table IV. The pattern of increased reporting of PMS up to three children but least PMS if there were four or more children persisted even when age and natural cycles were controlled (overall $\chi^2=15.1$, 6 d.f., $P=0.019$). In the pill-using group there was a similar distribution of family sizes and a similar increase in PMS from one to three children, although in this group of only 273 women this was not statistically significant.

Associations of Psychosocial Factors with reported PMS-status

Domestic circumstances

Women were categorised according to whether they were in a cohabiting relationship, or, if not, whether they were single or separated/divorced/widowed. This last category accounted for only 3% of nulliparous women and 8% of parous women and will not be considered further. Almost all parous women were cohabiting (91%) so there was very little scope to detect an association between cohabitation and PMS status in this group. For nulliparous women the cohabiting/single split was fairly even (50% and 46% respectively) and some association with PMS was found. In the subgroup under 25 years of age there was slightly more PMS among cohabiting than single women (45% v. 40%) but above this age the situation was reversed. For example, in the 25–29 year age group there was significantly more PMS among single women (61% v. 50%: $\chi^2=17.2$, 4 d.f., $n=643$, $P=0.0018$). This effect for those over 24 years persisted even when OC use and 'natural' cycles were controlled for, and it was most evident among women who were in other respects less likely to be reporting PMS (i.e. those who were using or had recently stopped oral contraception).

Employment

There was a statistically significant association between PMS-status and employment ($\chi^2=78$, 4 d.f., $P=0.0000$). PMS reporting was more prevalent among those working part-time (68% 'yes') than those with no paid job (61%)

TABLE IV
Family size and 'PMS-yes' reporting

Family size (no. of children)	*1 (n = 716)*	*2 (n = 1608)*	*3 (n = 587)*	*4+ (n = 186)*	*Total (n = 3097)*
% of total n	23	52	19	6	100
% 'PMS-yes'	65	68	73	62	68

or working full-time (56%). (The distribution of subjects across the three employment categories was 29%, 30% and 41% respectively). Once age and parous state were controlled for, the change in PMS prevalence across employment status persisted, although somewhat reduced, averaging out at an excess among part-time workers of about 5% if parous, or 9% if nulliparous and over 25 years of age.

Relationship

Although any woman who rated her relationship 'not at all happy' has been excluded from the analysis, the questionnaire allowed a range of other ratings: 'very happy' (57%), 'fairly happy' (37%) and 'not very happy' (6%). Among cohabiting women, PMS was related to the unhappiness of the relationship ($\chi^2=31.6$, 4 d.f., $P=0.0000$). In the parous subgroup of cohabiting women ($n=3024$), the prevalences of PMS were 65%, 69% and 72% respectively for the ratings detailed above and the corresponding figures for the nulliparous subgroup were 53%, 56% and 67% respectively ($n=1037$). The parous subgroup rated their relationships less favourably than the nulliparous: 54% v. 69% very happy, 6% v. 3% not very happy. On the other hand, among the 62% of single women who had a relationship (but were not cohabiting), there was no overall association between PMS reporting and their rating of the relationship.

In both 'PMS-yes' and 'PMS-maybe' groups, 77% of women thought that their partners were fairly or very understanding of their PMS, and these women were much more likely to rate their relationship as 'very happy'.

Stress

Although women undergoing a lot of 'unusual' stress have been excluded, the women included can be differentiated according to whether they report 'some' such stress (55%) or none (45%). There was an association between PMS-status and this stress ($\chi^2=23.3$, 2 d.f., $P=0.0000$) and this persisted when age and parity were controlled.

Psychosocial factor 'load'

Among nulliparous women, reporting of 'some' stress ranged from 53% in those under 25 years of age to 63% in women 35 years or older. The other psychosocial factors reported above are also related to age and the effects of the various factors have been found to persist when age was controlled. Thus the impact of age-related psychosocial factors may account for some of the age effect seen in Table III. In order to re-examine the age/natural cycles effects while controlling for psychosocial factors, a cumulative psychosocial factor 'load' was calculated as a count of the number of these factors applying in each case (i.e. unhappy cohabiting relationship, part-time employment, three children, 'some' stress and single state in those over 24 years of age). This 'load' is very strongly associated with age (parous: $\chi^2=75.2$, 12 d.f., $P=0.0000$; nulliparous: $\chi^2=24.8$, 6 d.f., $P=0.0004$, this latter χ^2 calculated excluding the contribution of the last item which is partly age-defined). There was, however, no overall association of current psychosocial 'load' with the duration of natural cycles (in non-pill using women), nor with an onset of PMS before the current interval of such cycles. This psychosocial 'load' could not be expected to be of equal sensitivity in the nulliparous and parous groups both because the potential variables assessed were not necessarily of comparable psychosocial impact in the two groups (and indeed one was applicable only to parous women) and because of the way in which component factors were ascertained independently in the parity groups. Thus when 'load', not comparable across parous state, was converted to a binary variable for use as a control factor, the cut-off points could be set separately in the two groups. For the sake of reasonable subgroup sizes, the cut-offs used were >0 for nulliparous and >1 for parous women.

Combined analysis

The pattern of PMS prevalences by age and natural cycles was examined in a combined analysis taking into account pill-use, parous state, prior PMS (i.e. starting before their current interval of natural cycles) and psychosocial 'load'. Some of the subgroup sizes became rather small, but in non-pill using women without prior PMS the numbers are on the whole adequate and the findings are reported.

Psychosocial 'load'

Since the factors comprising this 'load' were associated with higher prevalences of PMS one would expect lowered prevalences of PMS among women without psychosocial 'load'. Within subgroups based on natural cycles, however, this expected effect was only in the early and later years of such cycles ($\leqslant 2$ or $\geqslant 7$ years), as shown in Fig. 6 for nulliparous women over 24 years of age. Such a pattern was also evident in younger nullips and to a lesser extent in parous women.

Parous state

Here we consider not the *overall* parity effect, which could be affected by differences in sensitivity of the

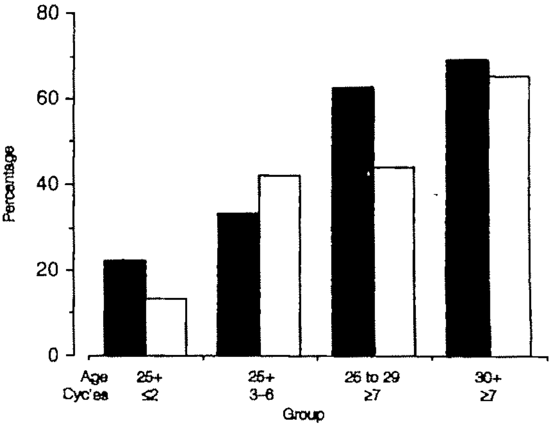


FIG. 6. Proportion of nulliparous women (non-pill-users, without prior PMS) reporting 'PMS-yes' by psychosocial 'load', age and duration of natural cycles (psychosocial load: none, $n=220$ (□); some, $n=424$ (■)).

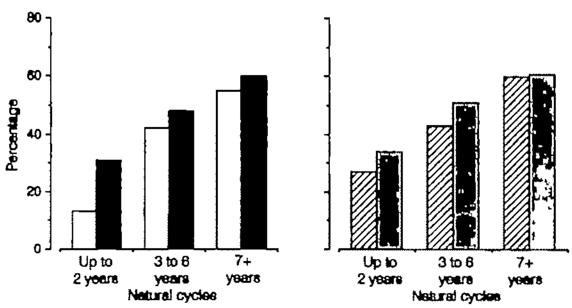


FIG. 7 Proportion of women (non-pill-users with no psychosocial 'load' or prior PMS) reporting 'PMS-yes' by duration of natural cycles: (a) comparing parous women (30–39 years, $n = 817$ (■)) with nulliparous ones (≥25 years, $n = 220$ (□)); (b) parous women only: post-pill ($n = 283$ (■)) v. post-birth ($n = 534$ (▨)).

control variable 'load' in the two parity groups, but variations in the size of the parity effect in relation to other variables. For example, what parity effect there is, is most evident in the early years of natural cycles. This is illustrated in Fig. 7*a*, comparing parous women (aged 30–39 years) and nulliparous ones (aged >25 years), grouped according to duration of natural cycles. In the early years, this effect was, in particular, attributable to those parous women who had not used oral contraception since their last birth, as shown in Fig. 7*b*. This is similar to what was reported above for the uncontrolled data.

Duration of natural cycles

This effect persists strongly and the increase in prevalence of PMS from under two to more than six years of cycles averages 30–40%. In women with no psychosocial 'load' the greatest increase in prevalence of PMS occurs after two years of natural cycles and this is most evident in nulliparous women (illustrated in Fig. 7*a*). On the other hand, among women with psychosocial stresses the greatest increase was after six years of natural cycles (illustrated for nulliparous women in Fig. 6, columns 3 and 5).

Age

Once psychosocial 'load', prior PMS and natural cycles have been controlled for, there is little age effect remaining except perhaps in nulliparous women with no 'load' and with longest duration of natural cycles (≥7 years) where there is a marked increase in PMS in those over 30 years of age (c.f. columns 6 and 8 of Fig. 6).

Discussion

Not all women with perimenstrual variation in mood or physical state will discern the cyclical nature of their symptoms, and of those who do, not all will deem themselves to be suffering from PMS. In our study the self-designated status of PMS sufferer by 61% of women has been substantially validated by their more detailed retrospective symptom reporting. We found PMS status to depend more on emotional than physical symptoms, and, in particular, on the number of severe mood symptoms pre-menstrually. However, it was striking that symptoms corroborative of PMS were also reported by the majority of women 'uncertain' about their PMS status and by a substantial minority of the 'no PMS' group.

There is a common belief, derived mainly from clinical or 'volunteer' experience (e.g. Hargrove & Abraham, 1982; Moos, 1968), that PMS is more prevalent among parous women and those around 30 years of age. It is somewhat surprising then that two recent population surveys found little evidence to support this: Andersch *et al* ($n = 1083$) found no change in severity of individual symptoms with parity, and only anxiety, a relatively uncommon pre-menstrual symptom, increased with age; and in the Hallman study ($n = 1852$), the overall multiple-regression analyses of the mental, somatic and total symptom scores found that they were unrelated to age, and that only the mental score was moderately related to parity. However, the latter survey also showed that self-designated PMS status *was* related to age, being most prevalent in the 25–38 year age groups. Furthermore, within the PMS group, the subgroup desiring treatment for their PMS (7.5% of entire sample) had a markedly higher proportion of parous women. This suggests that there are factors over and above pre-menstrual symptom experience which contribute to a woman's perception of herself as a PMS sufferer, or which increase her concern about the impact of it on her life, perhaps even to the extent of seeking treatment. An understanding of these factors would give insight into the aetiologies of PMS. In this paper, it is such self-perception as a sufferer of PMS that is under study.

Participation in our survey is likely to indicate concern about or at least interest in problems related to the menstrual cycle. This is confirmed by the markedly higher response rate in the 25–35-year age group, the low proportion of women (7%) who believed they did not suffer PMS (compared to 27% in Hallman's survey), and the considerably higher prevalence of severe symptom reporting, even allowing for different measurement methods (65% of our respondents reported pre-menstrual irritability to a 'severe' or 'very severe' degree compared with only 3% 'severe' in Hallman's PMS group). Thus, in this sample of women concerned about PMS, the association between self-designated PMS status and other factors is of particular interest. We have found striking associations between PMS status, duration of natural cycles and various combinations of psychosocial and demographic factors. Weaker but interesting associations have been found with oral contraceptive use and parity.

Natural cycles

The longer a woman has been experiencing natural menstrual cycles, unmodified by oral contraceptives and uninterrupted by pregnancy, the more likely she is to regard herself as a PMS sufferer. Further studies are required to replicate this potentially important finding and to identify the underlying mechanism. Is it a cumulative effect of repeated cycles, and if so is the mechanism mainly biological or psychological? In this regard, the interaction between stress and natural cycles is intriguing. It would appear that stress is strongly implicated in perception of PMS in the early and later years of a sequence of uninterrupted cycles but not around three to six years. At this time some other factor seems to be obscuring the effect of stress on PMS, possibly biological disturbance. Interestingly, it is women in the age range 25–35 years (already shown to be a time of increased concern about PMS) who are most likely to have been experiencing uninterrupted natural cycles for three to six years (Fig. 4).

On the other hand, if one considers the generally low prevalence of new perception of PMS in the first two years of natural cycles, there is suggestion that an interruption to natural cycles, in particular by recent oral contraceptive use, may exert some protective effect for a couple of years, either through the elimination of (real) menstrual cycles for a time, or through some slightly more lasting effect on the hypothalamic pituitary-gonadal axis.

Psychosocial factors

Our findings have highlighted the complex interactions of these factors with perception of PMS. For example, dissatisfaction with relationship was found to be a relevant factor for cohabiting but not non-cohabiting women, and the single state for nulliparous women over 25 years of age, but not for younger women. No particular psychosocial factor was of paramount importance in the group as a whole, and it would not be possible to control adequately for these diverse factors in studies which aim to assess PMS more thoroughly (e.g. by means of prospective ratings which would thus constrain the study to fairly small numbers).

As is common when psychosocial effects are being explored, the factors reported here are of low specificity and sensitivity, resulting partly from their indirect nature (e.g. it is not part-time work *per se* that is stressful, but rather some related condition(s), which may be present to a greater or lesser extent in all three 'employment' categories). Rather than continuing to utilise the standard demographic variables as an indirect assessment of underlying psychosocial stress, research efforts should be directed towards devising a summary measure of stress/dissatisfaction relevant to PMS. In this regard, it is striking that we obtained strong effects for 'unusual stress' and 'unhappy relationship' even though women with the most extreme ratings for these variables (and with even higher prevalence of PMS) had been excluded from the analysis. It would also be important that such a measure should ascertain health-related events so that their effects can be allowed for, along the lines of Woods *et al* (1982). Such a measure would be invaluable in surveys and also as a control variable in prospective studies.

Oral contraceptive use

As has been reported in a number of studies (Moos, 1969; Kutner & Brown, 1972), women using OCs, were less likely to report PMS, especially the nulliparous ones. However, both exacerbation of PMS and therapeutic effect have been reported for use of such contraceptives (Reid & Yen, 1981). Thus the strong association between PMS reporting and past OC-use among the younger women in our survey is of particular interest. In older women, discontinuation of the use of OCs often reflects either fears of long-term risks of steroidal contraception or a preference for sterilisation once family size is complete. In younger women, on the other hand, discontinuation is more likely to reflect adverse reactions to the OC. These findings are therefore compatible with previous reports that women with PMS are more likely to have experienced adverse effects on OCs (Dennerstein *et al*, 1984; Sanders *et al*, 1983). This is an issue of considerable importance in understanding the determinants of PMS. It also emphasises the point that in any population of long-term OC users, women with a tendency to PMS will have selected themselves out.

On the other hand, while one cannot conclude that OCs necessarily alleviate PMS, it is possible that their use, by women free of adverse reactions to them, affords some protection against the development of PMS. We found that among ex-pill-users, PMS was less often reported by women who had recently discontinued OCs (i.e. with shorter duration of natural cycles).

Parity

Women often date the onset or at least the worsening of PMS to a post-partum phase. While it is believed that pregnancy may be associated with biological changes conducive to PMS, transition to the parous state or increase in family size is undoubtedly also associated with psychosocial stress. In our survey the effect of parity was most evident in the early years of natural cycles (Figs 5 & 7*a*) in particular among women who had not used OCs since birth and who were closer to parturition (Fig. 7*b*). While this might be expected, in view of the psychosocial effect of a new baby, there is also the possibility of pre-menstrual recurrences of post-partum affective disorder. (In either case this 'proximity to parturition' effect would be working against the effect of duration of natural cycles.)

Overall though, the effect of parous state on the prevalence of PMS was only modest. However, looking within just the parous group, there was a marked increase in PMS prevalence with family size (up to three children). This would be compatible with either a cumulative psychosocial or biological effect of parity, providing we can account for the fact that the small proportion of women with four or more children (6%) were least likely to report PMS (controlling for age and duration of cycles) (Table IV). Given modern methods of family planning, it is likely that in most cases in this subgroup, the fourth (or subsequent) baby was planned, so the woman is likely to be happy in her mothering role. On the other hand, a proportion of women with smaller families may have decided to limit their families either because of a prior post-partum illness (for fear of another), or because they found parenting stressful or were experiencing other difficulties (e.g. marital), and any of these might be associated with subsequent development of PMS. (We have elsewhere reported an association between pre-menstrual mood change and earlier post-partum depression (Warner *et al*, 1990) and we have reported above an association between psychosocial stress and PMS.) Alternatively, they may have limited their family size because they were already suffering PMS and this made for difficulties in their mothering role. Thus there might be a selection out of PMS or of factors associated with PMS in women choosing to have large families. Greater insight into the relative contributions to the aetiology of PMS, of post-partum affective illness, biological effects of pregnancy, demands and commitment of parenting or other stresses would require a prospective study of women making the transisiton to the parous state.

Age

Once all of these factors are taken into account, there is very little evidence of any remaining age effect except in 'unstressed' nulliparous women with the longest duration of cycles. However, among nulliparous women reported stress 'load' increased with age, and in these older age groups there may also be at work other psychosocial factors not measured in our questionnaire. Overall, it seems that 'age effect' can be explained in terms of duration of current interval of natural cycles, psychosocial stress and prior opportunity to have discerned oneself as a PMS sufferer.

Conclusions

The most striking finding is the relationship between perception of PMS and duration of natural cycles, and the interaction of this association with psychosocial 'load'. There has been little research attention paid to the longitudinal course of PMS, and while the same is largely true of affective illness, Post *et al* (1986) have described a conditioning-sensitisation model to explain the longitudinal course of affective illness. They suggest the model may also apply where there are repeated endogenous events such as cyclical pre-menstrual dysphoria. In a 'sensitised' subject, subsequent episodes of dysphoria would be more easily triggered by lower levels of biological or psychosocial 'stressors'. Our findings could be consistent with such a model: if at about three years of uninterrupted natural cycles there is an interval of biological 'disturbance' causing pre-menstrual changes, it might also serve as a suitable conditioning phase for subjects vulnerable to sensitisation. In such subjects the PMS would be more likely to persist after the biological substrate had normalised. This would explain our finding that after six years of natural cycles there was a higher prevalence of PMS among those with psychosocial 'load' (or stressors), an effect not seen at three to six years of natural cycles. In addition, PMS may persist as a result of biological sensitivity, possibly interacting with psychosocial stressors.

Such a model also has great appeal for explaining some of the conflicting findings of PMS such as, at the biological conditioning stage of PMS there may well be detectable endocrine markers of PMS, whereas if a sample includes a substantial proportion of women in the later 'sensitised' but biologically normal stage it would be unlikely that any hormonal differences would be found between sufferers and controls. Similarly, the differential effect of ovulatory and anovulatory cycles might be evident only at a particular stage in the longitudinal course of PMS, and the association of PMS with predisposition to affective disorder may be found only in that subgroup of women whose PMS persists into the stage sensitised to psychosocial stress.

In a sensitised subject, behavioural changes are likely to be more marked and of longer duration. The findings reported in this paper are based on analysis in which PMS-status was an ordinal variable. In a further paper we will examine these associations more closely, using measures not only of severity of cyclical symptoms but also of different patterns of symptom change (Warner & Bancroft, in preparation). It may be, for example, that the associations with duration of natural cycles or with parity are most evident with certain types or timing of perimenstrual symptoms, and this may provide further insight into the longitudinal course and aetiology of PMS.

Acknowledgements

The authors wish to thank Anne Walker who piloted the method of symptom measurement and Lynn Williamson and Amanda Dixson for their assistance with the processing of the questionnaires.

References

Andersch, B., Wendestam, C., Hahn, L., *et al* (1986) Premenstrual complaints. I. Prevalence of premenstrual symptoms in a Swedish urban population. *Journal of Psychosomatic Obstetrics and Gynaecology*, **5**, 39–49.

Bancroft, J. & Backstrom, T. (1985) Premenstrual syndrome. *Clinical Endocrinology*, **22**,313–336.

Dennerstein, L., Spencer-Gardiner, C., Brown, J. B., *et al* (1984) Premenstrual tension: hormonal profiles. *Journal of Psychosomatic Obstetrics and Gynaecology*. **3**, 37–51.

Endicott, J. & Halbreich, U. (1982) Retrospective report of premenstrual depressive changes. Factors affecting confirmation of daily ratings. *Psychopharmacology Bulletin*, **18**, 109–112.

—— & —— (1988) Practical problems in evaluation. In *Contemporary Issues in Obstetrics and Gynaecology. Vol 2. The Premenstrual Syndrome* (ed. L. H. Gise), pp. 35–46. Edinburgh: Churchill Livingstone.

Hallman, J. (1986) The premenstrual syndrome – an equivalent of depression? *Acta Psychiatrica Scandinavica*, **73**, 403–411.

Hamilton, J. A. & Alagna, S. W. (1988) Toward a clinical research perspective. In *Contemporary Issues in Obstetrics and Gynaecology. Vol 2. The Premenstrual Syndrome* (ed. L. H. Gise), pp. 35–46. Edinburgh: Churchill Livingstone.

Hargrove, J. T. & Abraham, G. E. (1982) The incidence of premenstrual tension in a gynaecological clinic. *The Journal of Reproductive Medicine*, **27**, 721–724.

Hart, W. G., Coleman, G. J. & Russell, J. W. (1987) Assessment of premenstrual symptomology: a re-evaluation of the predictive validity of self-report. *Journal of Psychosomatic Research*, **31**, 185–190.

Kutner, S. J. & Brown, S. L. (1972) Types of oral contraceptives, depression and premenstrual symptoms. *Journal of Nervous and Mental Disorders*, **155**, 153–162.

Logue, C. M. & Moos, R. H. (1986) Perimenstrual symptoms: prevalence and risk factors. *Psychosomatic Medicine*, **48**, 388–414.

Miller, P., McC., Kreitman, N. B., Ingham, J. G., *et al* (1989) Self-esteem, life stress and psychiatric disorder. *Journal of Affective Disorders*, **17**, 65–75.

Moos, R. (1968) The development of a menstrual distress questionnaire. *Psychosomatic Medicine*, **3**, 853–867.

—— (1969) Assessment of psychological concomitants of oral contraceptives. In *Metabolic Effects of Gonadal Hormones and Contraceptive Steroids* (ed. K. A. Salhanik), pp. 676–705. New York: Plenum.

Post, R. M., Rubinow, D. R. & Ballenger, J. C. (1986) Conditioning and sensitisation in the longitudinal course of affective illness. *British Journal of Psychiatry*, **149**, 191–201.

Reid, R. L. & Yen, S. S. C. (1981) Premenstrual syndrome. *American Journal of Obstetrics & Gynecology*, **139**, 85–104.

Sanders, D., Warner, P., Backstrom, T., *et al* (1983) Mood, sexuality, hormones and the menstrual cycle. I. Changes in mood and physical state: description of subjects and methods. *Psychosomatic Medicine*, **45**, 487–501.

Schilling, K. M. (1981) What is a real difference? Content or method in menstrual findings. In *The Menstrual Cycle: Research and Implications for Women's Health, Vol. 2* (eds P. Komnenich, M. McSweeney, J. A. Moack, *et al*). New York: Springer.

Warner, P., Bancroft, J., Dixon, A. *et al* (1990) The relationship between perimenstrual mood change and depressive illness (submitted for publication).

Woods, N. F., Dery, G. F. & Most, A. (1982) Stressful life events and perimenstrual symptoms. *Journal of Human Stress*, **8**, 23–31.

*Pamela Warner, BSc, *Senior Research Officer, MRC Reproductive Biology Unit, 37 Chalmers Street, Edinburgh EH3 9EW*; John Bancroft, MD, FRCP, FRCPsych, *Clinical Consultant, MRC Reproductive Biology Unit, Edinburgh*

*Correspondence

British Journal of Psychiatry (1990), 157, 261–264

A Controlled Study of Psychological and Social Change after Surgical Gender Reassignment in Selected Male Transsexuals

CHARLES MATE-KOLE, MAURIZIO FRESCHI and ASHLEY ROBIN

Of two groups of 20 patients accepted for gender reassignment surgery, one was offered early operation and therefore had had surgery by follow-up two years later, while the second was still awaiting operation at two-year follow-up. Although the groups were similar initially, significant differences between them emerged at follow-up in terms of neuroticism and social and sexual activity, with benefits being enjoyed by the operated group.

Hoenig (1982) described transsexuals as:

> "persons who are born without physical abnormality in their genital organs . . . they are assigned to the opposite sex and reared in that sex. And yet in spite of all this and often in the face of fierce resistance by parents and others, they develop, usually from the earliest age, a paradoxical gender identity . . . [which is] permanent and unchangeable. Most transsexuals pursue their aim of physical sex change with fanatical fervour and the idea . . . appears to dominate their entire existence."

Roberto (1983), reviewing an extensive literature, concluded that:

> "the clinical definition of adult transsexualism is based on a composite set of characteristics . . . the belief that one is a member of the opposite sex . . . dressing and behaving in the opposite gender role . . . perceiving oneself as heterosexual although sexual partners are anatomically identical . . . repugnance for one's own genitals and the wish to transform them . . . and a persistent desire for conversion surgery."

Transsexualism is seen in both sexes but males present more commonly for treatment. While gender reassignment surgery was first described over 50 years ago (Abraham, 1931), the stimulus for the large-scale application of surgery came many years later (Hamburger *et al*, 1953) with the publicity surrounding the Jorgensen case. "As of the beginning of 1979 estimates of the number of adult Americans treated hormonally and surgically ranged from 3,000 to 6,000" while "as of mid-1978 approximately 40 centres in the western hemisphere offered surgical sex reassignment" (Harry Benjamin International Gender Dysphoria Association (IGDA), 1985). Despite a significant increase in the numbers treated since then, differences of opinion concerning outcome continue (Williams, 1987). A prospective controlled study of male transsexuals accepted for surgical reassignment is described here.

Method

Referrals to the Gender Identity Clinic at Charing Cross Hospital were routinely examined by a psychiatrist using a standard history form at first attendance and, if the patient proceeded to surgery, on acceptance for the surgical waiting-list. This form covered personal and family medical and psychiatric history, development, education, work record, social and sexual relationships, and the onset and progress of the patient's transsexualism.

At the same points in time a psychologist (CMK) administered the Crown Crisp Experiential Index (CCEI; Crown & Crisp, 1979), which is designed to measure different aspects of neurotic symptoms: free-floating anxiety (FFA), phobic anxiety (PHO), obsessionality (OBS), somatic anxiety (SOM), depression (DEP), and hysteria (HYS). CMK also administered the Bem Sex Role Inventory (BSRI; Bem, 1974, 1977, 1981), which sorts, in quantitative terms, self-assessments of 60 personality characteristics to define 'femininity' and 'masculinity' as separate entities.

Selection for surgery was decided by the standards of care recommended by the IGDA (1985) and included psychiatric assessment and treatment for a minimum of six months to confirm diagnosis, exclude psychosis, and assist in change of gender role. Criteria included a persistent wish (at least two years) to change gender, and demonstrable ability to live adequately in the chosen gender role for at least a year by working and becoming self-supporting in that role. Randell (1971) in addition suggests that "the patient should be better adjusted in the role they desire than the role they had left . . . [and] must be willing to accept the limitations."

In normal practice it was unusual for a patient to be referred for surgery in under two years and psychiatric surveillance continued for the two to three years patients spent on the waiting-list, as well as two years post-operatively. The mean time, therefore, from first attendance to surgery, for the minority of referrals that finally achieved reassignment, was about five years. All patients were treated using a single-stage operation including penectomy, orchidectomy and the construction of a neo-vagina.

Within three months of acceptance onto the operation waiting-list, 20 alternate patients from a serial list of 40 (mean age 32.5 years, range 21–53 years) were offered early surgery while their alternate was dealt with routinely and thus awaited operation at the usual time. All 40 patients were re-examined after two years by a psychiatrist and psychologist using the same test battery as before. At this time the 'advanced' operation group (group A) had had surgery about one year and nine months previously, while the 'routine' group (group R) was still awaiting surgery.

Results

Comparison of groups on acceptance for surgery

Selection of alternate waiting-list patients should produce satisfactory random groups, and in fact the two groups did have similar personal family and psychiatric histories (Table I). Moreover, the groups had been selected for operation on the basis of their psychiatric stability. The groups were also similar in their family constellations, and as far as first-degree relationships, family preferences and identifications were concerned. Both groups were closer to 'mother' and identified with her rather than with other family members. (In this context Edgerton *et al* (1982) point out that the mothers of male transsexuals accept the patient's condition in most cases while the fathers do not.) The two groups were also similar in employment status and in their level of activity as far as a wide variety of social interests was concerned.

In both groups cross-dressing emerged at around the age of six years. In sexual fantasy the overwhelming majority of patients saw themselves to be in the female gender role but in sexual practice were asexual, probably because during their assessment they had been treated with oestrogens for a significant time.

The mean BSRI scores of the two groups were not significantly different. Femininity scores (means: group A, 4.91; group R, 5.06) exceeded masculinity scores (means: group A, 4.47; group R, 4.57) in both groups and approximated to Bem's reference group of North American females (means: femininity, 5.05; masculinity, 4.79) rather than to North American males (means: femininity, 4.59; masculinity, 5.12). Finally, there were no significant differences on any measure in the CCEI (Table I). In summary therefore, at the time of acceptance for surgery groups A and R resembled each other closely in their histories and on all measures employed.

TABLE I

Characteristics of two groups of transsexuals accepted for gender reassignment surgery, before surgery

	Booked for early surgery (group A) (n = 20)	*Normal waiting time for surgery (group R) (n = 20)*
Psychiatric history in first-degree relations	4	6
Group mean number of childhood neurotic traits	7	8.5
Psychiatric treatment for non-gender problems	6	5
History of parasuicide	1	2
CCEI test scores		
Free-floating anxiety	5.85	4.75
Phobic anxiety	5.15	4.50
Obsessionality	6	6.10
Somatic anxiety	3.85	3.45
Depression	3.65	3.75
Hysteria	4.95	5.85

Changes over two years from entry to the surgical waiting-list

Applying the McNemar test for the significance of changes (Siegel, 1956), ratings showed a significant increase in social and sexual activity in group A but no change in group R (Table II). Operated patients became more active as far as all types of sport were concerned and more active socially, with both family and friends. As working in the cross-gender role was among the criteria for acceptance for surgery, the majority of both groups were employed when accepted for the waiting-list. However, group A remained

TABLE II

Significant changes during two-year follow-up in social, sexual and work activity for transsexuals post-operatively (group A) or awaiting operation (group R)

	More active	*Same*	*Less active*	*McNemar test* (χ^2)	P
Sport in company					
A	16	2	2	9.4	<0.01
R	1	16	3	<1	NS
Sport practised alone					
A	14	4	2	7.6	<0.01
R	1	16	3	<1	NS
Visits to family, friends, etc.					
A	15	3	2	8.5	<0.01
R	3	14	2	<1	NS
Dancing					
A	15	3	2	8.5	<0.01
R	1	15	4	<1	NS
Eating out					
A	15	3	2	8.5	<0.01
R	1	15	4	<1	NS
Visits to pubs, etc.					
A	12	6	2	5.8	<0.02
R	3	13	4	<1	NS
Sexual interest					
A	16	4	0	14.1	<0.001
R	0	17	3	1.3	NS
Work record					
A	0	19	1	<1	NS
R	0	14	6	4.2	<0.05

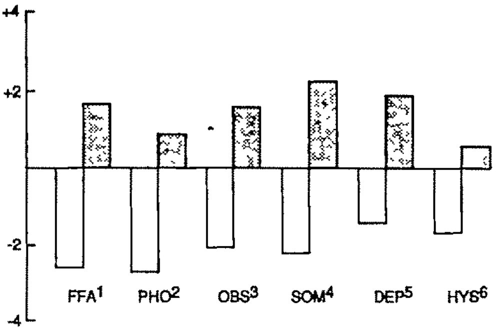


Fig. 1. Change in mean score of the operated (▨) and unoperated (☐) groups on each measure of the CCEI (1. $F=3.56$, d.f. 1,38, $P<0.05$; 2. $F=12.98$, d.f. 1,38, $P<0.005$; 3. $F=20.96$, d.f. 1,38, $P<0.001$; 4. $F=20.23$, d.f. 1,38, $P<0.001$; 5. $F=15.35$, d.f. 1,38, $P<0.001$; 6. $F=7.83$, d.f. 1,38, $P<0.01$).

in work while group R was significantly more often unemployed at follow-up.

Significant differences were also seen *between* the groups at follow-up, with group A more active than group R on all items mentioned above except social drinking and work. No differences were seen on the other items of social activity surveyed – working in the home and garden, cinema and theatre attendance, spectator sport, club membership, church attendance, gambling, reading, watching television, etc.

Scores on the BSRI did not change significantly in either group but the direction of change was, as previously noted (Mate-Kole *et al*, 1988), with masculinity scores increasing after surgery. Finally, all scores on the CCEI increased in the unoperated group and fell in the operated group (Fig. 1). The difference between the groups' change in mean score was highly significant for PHO, OBS , SOM, DEP, and HYS, but just failed to reach significance for FFA.

Discussion

An earlier follow-up of patients treated with gender reassignment surgery at Charing Cross Hospital (Randell, 1971) reported that 32 of 37 patients made an 'excellent', 'good' or 'fair' adjustment. One patient's adjustment was described as being 'poor' and four were 'worse' after operation. Two patients committed suicide. Edgerton *et al* (1982), describing a follow-up of 78 male transsexuals, reported that "every patient has continued to express satisfaction with the gender reassignment" although "over 15% indicated varying degrees of disappointment with the degree of surgical reconstruction". In subjective reports the patients described themselves as happier and more relaxed. Lundstrom *et al* (1984), summarising two previous reviews of the literature, noted that 71–88% of male transsexuals had 'satisfactory' outcomes, 8–10% had 'unsatisfactory' outcomes, while in addition 2% committed suicide.

Adverse prognostic factors include a poor surgical result (although the relationship is a complex one affected by the patient's perceptions), personal and social instability, increasing age, and secondary transsexualism (the incidence of which is directly related to age). In addition, Walinder *et al* (1978), from a study of patients seeking reversal of reassignment, included "physical characteristics inappropriate to the new sex role" such as "heavy bone structure, large hands, athletic muscle development, coarse features, for example, profuse body hair" as adverse prognostic factors. Lothstein (1984) specifically reviewed "psychological testing" in transsexuals and concluded that male patients became psychologically stabilised once they lived out and enacted a cross-gender role, and became 'more stable' after hormones and surgical interventions.

Meyer & Reter (1979) were more sanguine in their conclusions, based on a follow-up of only 50% of a cohort, and a comparison of unoperated patients accepted as applicants for surgery, some of whom later proceeded to surgery, with a group of operated patients. The authors were clear that the unoperated individuals were "not a rigorous control group" but "the only available approximation to it". The authors noted differences in the racial distribution of the groups and that the follow-up in the operated group was two and a half times longer than in the unoperated group. Psychiatric contact was less frequent after operation than in the unoperated group and tended to be concerned with administrative matters and 'understanding'. Outcome was measured using an idiosyncratic scale which reduced legal, economic, domestic and psychiatric adjustment categories to a single digit, and initial scores appear to have been derived retrospectively. The range of adjustment scores was twice as great in the operated group compared with the unoperated. No differences were seen in the change of score between groups, although all groups showed better adjustments over time. Contrary to most other opinion, the operated group which had, in the main, completed a period of living in the cross-gender role was "slightly more distressed than the unoperated group" and the period of assessment was not regarded as 'treatment' but merely "the passage of time". The final conclusion of these authors was that transsexual applicants 'self-select' into two groups, for or against surgery; that in terms of social rehabilitation both groups make equal progress over time, but that subjective satisfaction is enjoyed by those who undertake surgical reassignment. The conclusion undoubtedly invalidates the aim of the study in that it admits the comparison of dissimilar groups. Surgery has never been proposed for those who do not wish it and the demand for it has often been regarded as inherent in the clinical definition of transsexualism (Hoenig, 1982).

Our own study deals only with those who 'self-select' for reassignment, persist with the demand for it over a long time, and are accepted for surgery after the rigid exclusion of known adverse factors – psychiatric instability (either personality disorder or psychosis) and an inability to maintain a better adjustment in the original gender role. The study is prospective and uses a validated measure of neurotic symptoms while examining social and sexual activity in explicit areas. Patients who achieve reassignment during the same period are seen to improve significantly as far as neurotic symptoms are concerned and to become more socially active in comparison with similar patients not yet reassigned.

Transsexualism is classified in ICD–9 (World Health Organization, 1978) as a 'mental disorder' (302.5) and gender reassignment surgery is a cosmetic procedure, in some cases allowing alternative sexual practice. The relief achieved with gender reassignment surgery is primarily as far as dysphoria and its consequences are concerned, and is similar to that achieved in other cases of cosmetic surgery (Hay & Heather, 1973; Robin *et al*, 1988). In selected cases it may be seen as a valid method of treatment.

Acknowledgements

The authors wish to thank Mr Peter Philip and Mr James Dalrymple for allowing access to patients under their care, Professor J. B. Copas, University of Birmingham, for advice on statistical method, Professor Steven Hirsch for constructive criticism, and Pauline Carty for invaluable secretarial assistance.

References

Abraham, F. (1931) Genitalumwandlung an zwei maennlichen transvestiten. 2. *Sexualwissenschaft*, **18**, 223–236.

Bem, S. (1974) The measurement of psychological androgyny. *Journal of Consulting and Clinical Psychology*, **42**, 155–162.

—— (1977) On the utility of alternative procedures for assessing psychology androgyny. *Journal of Consulting and Clinical Psychology*, **45**, 196–205.

—— (1981) *Bem Sex Role Inventory. Professional Manual*. Palo Alto: Consulting Psychologists Press.

Crown, S. & Crisp, A. (1979) *Manual of the Crown Crisp Experiential Index*. London: Hodder & Stoughton.

Edgerton, M. T., Langman, M. W., Schmidt, J. S., *et al* (1982) Psychological considerations of gender reassignment surgery. *Clinics on Plastic Surgery*, 9, 355–366.

Hamburger, C., Sturup, G. K. & Dahl-Iverson, E. (1953) Transvestism. Hormonal psychiatric and surgical treatment. *Journal of the American Medical Association*, **152**, 391–396.

Harry Benjamin International Gender Dysphoria Association (1985) Standards of care: the hormonal and surgical sex reassignment of gender dysphoric persons. *Archives of Sexual Behaviour*, **14**, 79–90.

Hay, G. G. & Heather, B. B. (1973) Changes in psychometric test results following cosmetic nasal operations. *British Journal of Psychiatry*, **122**, 89–90.

Hoenig, J. (1982) Transsexualism. In *Recent Advances in Clinical Psychiatry, Vol. 4*, (ed. K. Granville-Grossman). Edinburgh: Churchill Livingstone.

Lothstein, L. M. (1984) Psychological testing with transsexuals: a 30-year review. *Journal of Personality Assessment*, **48**, 500–507.

Lundstrom, B., Pauly, I. & Walinder, J. (1984) Outcome of sex reassignment surgery. *Acta Psychiatrica Scandinavica*, **70**, 289–294.

Mate-Kole, C., Freschi, M. & Robin, A. (1988) Aspects of psychiatric symptomatology at different stages in the treatment of transsexualism. *British Journal of Psychiatry*, **152**, 550–553.

Meyer, J. K. & Reter, D. J. (1979) Sex reassignment: follow-up. *Archives of General Psychiatry*, **36**, 1010–1015.

Randell, J. (1971) Indications for sex reassignment surgery. *Archives of Sexual Behaviour*, **1**, 153–161.

Roberto, L. G. (1983) Issues in diagnosis and treatment of transsexualism. *Archives of Sexual Behaviour*, **12**, 465–473.

Robin, A. A., Copas, J. B., Jack, A. B., *et al* (1988) Reshaping the psyche – the concurrent improvement in appearance and mental state after rhinoplasty. *British Journal of Psychiatry*, **152**, 539–543.

Siegel, S. (1956) *Non-parametic Statistics for the Behavioural Sciences*. London: McGraw-Hill.

Walinder, J., Lundstrom, B. & Thuwe, I. (1978) Prognostic factors in the assessment of male transsexuals for sex reassignment. *British Journal of Psychiatry*, **132**, 16–20.

Williams, G. (1987) Gender reassignment today. *British Medical Journal*, **295**, 671, 1348.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD–9). Geneva: WHO.

*Charles Mate-Kole, *formerly Research Psychologist, Charing Cross and Westminster Medical School, London, now Department of Psychology, Camp Hill Hospital, 1763 Robie Street, Halifax, Nova Scotia B3H 3G2, Canada*; Dr M. Freschi, *Consultant Psychiatrist, Ealing District Hospital*; Ashley Robin, *Honorary Consulting Psychiatrist, Charing Cross Hospital, London*

**Correspondence*

British Journal of Psychiatry (1990), 157, 265–268

Clinical Features of Patients Attending a Gender-Identity Clinic

ALISTAIR BURNS, MICHAEL FARRELL and JEREMY CHRISTIE BROWN

Of 106 patients attending a gender-identity clinic, 73% satisfied DSM–III criteria for transsexualism. These DSM–III positives had a significantly younger age of onset and were less likely to experience sexual arousal with cross-dressing than those who did not satisfy DSM–III criteria. They were also significantly more likely to fulfil a definition of 'core transsexualism'. 'Core transsexualism' may represent a subgroup within DSM–III criteria for transsexualism and its defining features in this study were an early age of onset, low sexual activity, lack of sexual arousal with cross-dressing and homosexual orientation. Of the whole sample, 23% were referred for gender reassignment surgery, of whom 100% were DSM–III positive and 60% were 'core transsexuals'.

In 1953, Benjamin coined the word 'transsexualism' to describe men and women who had the subjective experience of wishing to be of the opposite sex and who sought treatment to this end. In the same year, the surgical removal of the male genitals in order to make a man as much like a woman as possible was reported (Hamburger *et al*, 1953). Since then, clinics have been set up for the assessment and support of these individuals and the media have shown a mixture of curiosity and revulsion about such 'sex changes'. Recently, there has been debate as to the exact status of these patients (Snaith, 1987) and, at a time of financial hardship for the National Health Service, the performance of sex-change operations has been criticised (Williams, 1987). Transsexualism is the extreme expression of a spectrum of gender dysphoria, a disturbance of gender identity (Dolan, 1987). Gender dysphoria is defined as a persistent sense that one's gender identity is in discord with anatomical sex, and may be accompanied by gender-discordant behaviour (e.g. cross-dressing).

There are various criteria by which transsexualism can be defined, the most widely recognised being the DSM-III criteria (American Psychiatric Association, 1980) which consist of: (a) a sense of discomfort and inappropriateness about one's anatomical sex; (b) a wish to be rid of one's own genitals and to live as the other sex; (c) that the disturbance has been continuous for at least two years and not just related to periods of stress; and (d) that the condition is not due to other mental or physical disorders, such as schizophrenia and intersex or genetic abnormality. These broad criteria do not include features which most clinicians consider important in the diagnosis, such as age of onset, presence or absence of sexual arousal with cross-dressing and a dislike of secondary sex characteristics. Those patients showing these additional features are referred to in the literature as 'core', 'true' or 'primary' transsexuals, although no fixed criteria for them have been agreed.

For the past eight years, a clinic for the assessment of such patients has been in operation at the Maudsley Hospital under the direction of one of us (JCB). The clinic is primarily a general psychiatric clinic and subjects are seen as part of the routine out-patient workload. Referrals are not limited to a defined catchment area. In an attempt to identify the characteristics of patients presenting with gender dysphoria, a retrospective case-note study of the patients referred to the clinic since its inception was performed. The findings are presented and discussed in relation to transsexualism.

Method

The case notes of patients who had attended the clinic were subjected to a standardised assessment.

An attempt was made to assess the age of onset in each case. This was defined as the age at which gender dysphoria and/or cross-gender behaviour occurred which was related to the presenting problem. The age of 13 years was taken as an arbitrary dividing line between 'early' and 'late' onset cases because it was likely to discriminate between pre- and post-pubertal onset.

The definition of sexual orientation in relation to transsexualism is open to confusion. The terms 'homosexual' and 'heterosexual' were used to indicate sexual preference based on the subject's anatomical sex at birth, i.e. a male to female transsexual was regarded as homosexual if a preference for men was expressed. While recognising the apparent paradox, this definition was used for the sake of consistency. Bisexuality referred to a combination of homosexual and heterosexual desire. Sexual activity was rated independently from the above – low activity being defined as less than one sexual contact per year and/or masturbatory frequency of less than six per year.

The patients were divided into those who did and did not satisfy DSM-III criteria for transsexualism. 'Core transsexualism' was considered to be present if: (a) the age of onset was before puberty; (b) the adoption of the cross-gender role was without sexual arousal; and (c) a dislike of secondary sexual characteristics was present (the last criterion, however, proved difficult to measure).

An estimate of associated psychiatric morbidity was made by recording if there was a history of overdose of drugs, self-mutilation or alcohol misuse (not in the setting of a depressive illness).

Results

A total of 106 case notes were reviewed. There were 83 men (mean age 34.3 years) and 23 women (mean age 26.6 years). Of the sample, 46% had been referred from another psychiatrist, 31% from general practitioners (GPs) and 26% from other sources such as social workers and transsexual self-help groups. Age of onset was determined with confidence in 87 subjects; just over half of these had an onset under the age of 13 years. In those cases where there was doubt, allocation to onset before or after puberty was done by discussion. Sexual activity was rated in 90 subjects. Significantly more males were heterosexual than females (45% compared with 13%; $P<0.001$, χ^2 test) and bisexuality was exclusively a male attribute (16%). Low sexual activity was described in 35% with an approximately equal sex prevalence. Sexual arousal with cross-dressing was confined to men, occurring in 36%. A history of drug overdose, deliberate self-harm (no cases of mutilation of the sex organs were recorded) or alcohol misuse was present in 39% (42% of men and 27% of women). No physical abnormalities suggestive of intersex were noted.

The group was divided into those who satisifed DSM-III criteria for transsexualism (DSM-III positive) and those who did not (DSM-III negative; Table I). There was no difference in the mean age of each group. Those who were DSM-III positive were more likely to have an onset before 13 years old, not to experience sexual arousal with cross-dressing and consequently to be categorised as 'core transsexuals'. They were also more likely to be referred for surgery. None of these features are DSM-III criteria. Of the 29 DSM-III-negative subjects, two had a schizophreniform psychosis, one had dysmorphophobia for the penis, six had disturbances of sexual orientation (mainly homosexual transvestites), seven were depressed, six had personality disorders and seven had no classifiable mental disturbance.

Table II shows those satisfying DSM-III criteria for transsexualism, divided into those with and without features of core transsexualism (core positive and negative respectively). The only significant difference between the two groups was a lower age of onset in the core positives, which was clearly a consequence of the definition used. However, although not statistically significant, a higher proportion of the core-positive group showed certain characteristics that were not part of the definition used: homosexual orientation and low sexual activity (Table II).

Table III outlines the characteristics of those referred and not referred for gender-reassignment surgery. A total of 23% were referred for surgery – there was no sex difference among these. The majority were homosexual ($P<0.05$) and had features suggestive of 'core transsexualism' ($P<0.05$).

TABLE I

Characteristics of patients who did and did not satisfy DSM-III criteria for transsexualism (DSM-III positive and negative respectively)

Characteristics	*DSM-III positive (n = 77)*	*DSM-III negative (n = 29)*
Sex: male/female	58/19	25/4**
Mean age: years (s.d.)	32.6 (12.3)	32.9 (12.1)
Age of onset		
<13	54	4
>13	20	9*
unknown[1]	3	16
Sexual orientation		
heterosexual	27	14
homosexual	29	7
bisexual	9	4**
unknown[1]	12	4
Low sexual activity		
yes	29	7
no	42	21**
unknown[1]	6	1
Sexual arousal with cross-dressing		
present	1	6
absent	69	16*
unknown[1]	7	7
Core transsexualism		
present	35	0
absent	42	29*
Referred for surgery		
yes	24	0
no	53	29*

*$P<0.001$, **NS at $P<0.05$ (χ^2 test).

1. Insufficient information in case notes.

Table IV describes the specificity and sensitivity of DSM-III and core transsexualism for predicting referral for surgery. DSM-III criteria predicted all those referred (100% sensitivity) but also included many who were not referred (reflected in a low specificity, 35%). Core transsexualism had a lower sensitivity (60%) but a higher specificity (75%), i.e. it contained far fewer who were not referred for surgery but missed out some who were referred.

Discussion

Transsexualism is rare. The exact prevalence remains uncertain but one UK study suggested a prevalence of 1:40 000 in men and 1:110 000 in women (Hoenig & Kenna, 1974). A more recent study (Eklund *et al*, 1988) found a prevalence rate in the Netherlands of 1:18 000 for men and 1:54 000 for women. It was postulated that a lower threshold for seeking treatment may be responsible for this higher prevalence. A general psychiatrist is unlikely therefore

TABLE II

Characteristics of patients satisfying DSM-III criteria for transsexualism, with and without features of 'core transsexualism' (core positive and negative respectively)

Characteristics	*Core positive (n=35)*	*Core negative (n=42)*
Sex: male/female	25/10	33/9**
Mean age: years (s.d.)	32.2 (12.7)	33.0 (12.0)
Mean age of onset: years (s.d.)	7.7 (2.8)	14.5 (9.5)
Age of onset: years		
<13	33	21
>13	2	18*
unknown[1]	0	3
Sexual orientation		
heterosexual	9	18
homosexual	17	12**
bisexual	2	7
unknown[1]	7	5
Low sexual activity		
yes	16	13
no	15	27**
unknown[1]	4	2
Sexual arousal with cross-dressing		
present	0	7
absent	33	34**
unknown[1]	2	1
Referred for surgery		
yes	15	9
no	19	30**
unknown[1]	1	3

$^*P<0.001$, **NS at $P<0.05$ (χ^2 test).

1. Insufficient information in case notes.

TABLE III

Characteristics of patients fulfilling DSM-III criteria for transsexualism, referred and not referred for reassignment surgery

Characteristic	*Referred (n=24)*	*Not referred (n=53)*
Mean age: years (s.d.)	29.6 (9.2)	34.0 (13.3)
mean age of onset: years (s.d.)	11.4 (7.4)	11.3 (8.2)
Male/female	17/7	41/12
Age of onset		
<13 years	16	37
>13 years	7	13**
unknown[1]	1	3
Sexual orientation		
heterosexual	4	22
homosexual	12	17*
bisexual	4	5
unknown[1]	4	9
Low sexual activity		
yes	11	18
no	11	31**
unknown[1]	2	4
Sexual arousal with cross-dressing		
present	1	9
absent	21	42**
unknown[1]	2	2
'Core' transsexualism		
present	15	20
absent	9	33*

$^*P<0.05$, **NS at $P<0.05$ (χ^2 test).

1 Insufficient information in case notes.

TABLE IV

Sensitivity and specificity of DSM-III and 'core transsexualism' to predict referral for surgery

	Clinical criteria	
	DSM-III	*Core transsexualism*
Sensitivity[1]	100%	60%
Specificity[2]	35%	75%

1. Percentage of all cases referred for surgery who satisfied the clinical criteria of DSM-III or core transsexualism.
2. Of all cases satisfying each clinical criteria, the percentage reflects those referred for surgery.

to see more than one or two cases in a lifetime. A gender dysphoria clinic has a useful role in the assessment of such patients in a general psychiatric setting. In addition to assessment, the clinic has a supportive role for transsexuals who are, generally speaking, required to live and work as a member of the opposite sex for two years before they would be considered for referral for reassignment surgery.

The male:female sex ratio in this sample (3:1) is similar to that of other studies (Pauly, 1968; Randell, 1959; Ecklund *et al*, 1988). Although sexual orientation does not appear in the diagnostic criteria for transsexualism, clincial experience is that homosexuality (that is, sexual preference for people of the anatomical sex at birth as the subject) is commoner. The prevalence of heterosexuality reported in the literature is between 28% and 56% in males and between 0% and 13% in females (Dixen *et al*, 1984; Sorensen & Hertoft, 1982) compared with the prevalence in this study of 45% and 13% respectively. Bisexuality was found exclusively in men. Significantly more patients with homosexual orientation were referred for surgery which may represent predetermined bias at referring only 'core cases'. Sexual arousal with cross-dressing did not occur in women but was present in 36% of men. This concurs with other studies which have suggested there is a subgroup of transsexual men with fetishistic tendencies (Freund *et al*, 1982).

In this sample, 73% satisfied DSM-III criteria for transsexualism. This was significantly associated with a younger age of onset, lower sexual arousal with cross-dressing and thus with the present concept of 'core transsexualism' as well as with increased likelihood of referral for surgery. Concepts not included in the DSM-III diagnosis of transsexualism, yet significantly associated with it, included an age of onset of less than 13 years and the absence of sexual arousal with cross-dressing.

There was a history of self-harm in 39% of the present sample which is marginally more than in other studies (e.g. Dixen *et al*, 1984). Although commoner in males (see Dixen *et al*, 1984), it did not differ between those referred or not referred for surgery.

Referral for surgery was significantly associated with both a diagnosis of core transsexualism and a DSM-III diagnosis of transsexualism. No difference in anatomical sex or age of onset was found between the two groups.

In summary, the present study shows that the majority of patients attending a gender dysphoria clinic satisfied DSM-III criteria for transsexualism and those who did not had a variety of other psychiatric diagnoses. It suggests that a discrete syndrome exists along the lines of DSM-III but also associated with younger age of onset, lack of sexual arousal with cross-dressing and the presence of 'core transsexualism'. The concept of 'core transsexualism' has a firm basis in clinical experience and appears to represent a subgroup within DSM-III. In this study, its defining characteristics were found to be an early age of onset, lack of sexual arousal with cross-dressing, homosexual orientation and low sexual activity. It is a better predictor for referral for surgery than DSM-III criteria. No physical abnormalities which could have contributed to the transsexual syndrome were found in this sample and these must be considered rare. Although the present sample may be atypical in many ways (e.g. in mode of referral), their basic characteristics are similar to other reports (e.g. Pauly, 1968; Dixen *et al*, 1988). Further studies of the phenomenology of transsexualism are required and need to be linked to the evaluation of the outcome of surgical intervention.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Benjamin, H. (1953) Transvestism and transsexualism. *International Journal of Sexology*, **7**, 12–14.

Dixen, J. M., Maddever, H., Van Maasdam, J., *et al* (1984) Psychosocial characteristics of applicants evaluated for surgical gender reassignment. *Archives of Sexual Behaviour*, **13**, 269–276.

Dolan, J. (1987) Transsexualism: syndrome or symptom? *Canadian Journal of Psychiatry*, **32**, 666–673.

Eklund, P. L. E., Gooren, L. J. G. & Bezemer, P. D. (1988) Prevalence of transsexualism in the Netherlands. *British Journal of Psychiatry*, **152**, 638–640.

Freund, K., Steiner, B. W. & Chan, S. (1982) Two types of cross gender identity. *Archives of Sexual Behaviour*, **11**, 49–63.

Hamburger, C., Sturup, G. & Dahl-Iverson, E. (1953) Transvestism. *Journal of the American Medical Association*, **152**, 391–396.

Hoenig, J. & Kenna, J. C. (1974) The prevalence of transsexualism in England and Wales. *British Journal of Psychiatry*, **124**, 181–190.

Pauly, I. B. (1968) The current status of change of sex operation. *Journal of Nervous and Mental Disease*, **147**, 460–471.

Randell, J. (1959) Transvestism and transsexualism. *British Medical Journal*, *ii*, 1448–1452.

Snaith, P. (1987) Gender reassignment today. *British Medical Journal*, **295**, 454.

Sorensen, T. & Hertoft, P. (1982) Male and female transsexualism: the Danish experience with 37 patients. *Archives of Sexual Behaviour*, **11**, 133–155.

Williams, G. (1987) Gender reassignment today. *British Medical Journal*, **295**, 671.

*Alistair Burns, MRCP, MRCPsych, MPhil, *Honorary Senior Registrar in Psychiatry, Institute of Psychiatry, de Crespigny Park, Denmark Hill, London SE5*; Michael Farrell, MRCP, MRCPsych, *Senior Registrar in Psychiatry, Maudsley Hospital, Denmark Hill*; Jeremy Christie Brown, FRCP, FRCPsych, *Consultant Psychiatrist, Maudsley Hospital*

*Correspondence

British Journal of Psychiatry (1990), 157, 269–272

Collected and Neglected: Are Oxford Hostels for the Homeless Filling up with Disabled Psychiatric Patients?

P. GARETY and R. M. TOMS

> "*Objective* – To assess the severity of psychiatric symptoms among residents of hostels for homeless people. *Design* – Survey of residents in two hostels in Oxford, comprising three weeks of background fieldwork, a demographic questionnaire, and rating behaviour over two weeks with a behavioural rating scale (REHAB) and mental state with the brief psychiatric rating scale. *Setting* – Two hostels for homeless people in Oxford. *Subjects* – 146 Medium to long term residents, of whom 48 were selected by hostel workers by the following criteria: continuous residence for at least two months, signs of persistent severe mental disability, and difficulty in coping independently in the community. Two subjects died during the study; three (previously long term psychiatric inpatients) declined to be assessed on the psychiatric scale. *Main outcome measure* – Behavioural disturbance and mental state. *Results* – Only a third of the total sample had been born in Oxfordshire. Subjects had been accepted into the hostel either by arrangement with the local psychiatric service (22) or straight off the streets (26); 43 had had a previous (non-drug related) psychiatric admission. Subjects were significantly more likely than other residents to have spent longer (>80 weeks) in a hostel in the past three years ($p<0\cdot02$). With reference to norms for deviant behaviour, the 46 subjects assessed showed considerable deviant behaviour (average weekly scores: 0 (11 subjects), 1 (14), 2–3 (16), and $\geqslant 4$ (5)) not significantly different from that expected in moderately to severely handicapped psychiatric inpatients ($\chi^2=1\cdot3$, $df=3$, $p>0\cdot7$); 22 had scores equivalent to those in most severely handicapped inpatients. Of the 43 subjects assessed with the psychiatric rating scale, 16 had symptoms of neurosis, 29 of florid psychosis, and 32 of a deficit state. Symptoms of deficit state were positively correlated with ratings of low social activity on the behavioural scale (Spearman's rank correlation coefficient $0\cdot30$, $p=0\cdot03$). *Conclusions* – Hostels are having to care for long term severely affected psychiatric patients discharged into the community. The suitability of the services offered to such subjects should be assessed."

The summary quoted above is from an article by Marshall (1989). The present authors were invited to comment upon the study.

P. Garety

Concern that the policy of deinstitutionalisation is making a significant contribution to the numbers of homeless people is now widespread. On 31 October 1989, the London *Evening Standard* published an emotive and potentially inflammatory article in which psychiatric patients were depicted as uniformly dangerous, deranged, and deprived, even of a place to sleep. Vignettes of current in-patients (who by venturing onto the local streets were assumed to be homeless) were intermingled with true and distressing accounts of deprivation, and frank fiction. Such articles are not uncommon, and reflect awareness of a real problem. However, it is of the utmost importance to gather accurate data about the homeless mentally ill, and in the pursuit of this, Dr Marshall's paper is an important and timely contribution to the literature.

In this study, hostel staff identified approximately one-third of a total population of two shelters for the homeless as suffering from a persistent and severe mental disability. Using a brief symptom rating scale (the BPRS), Marshall found approximately 90% to be currently floridly psychotic, or showing symptoms of a defect state. In addition, and more unusually, he assessed the functioning of the subjects: the level of disability and behavioural disturbance, as assessed by the REHAB scale, was high – at a similar level to that found in long-stay psychiatric wards for the moderately to severely disturbed.

Nearly all the subjects had been in contact with psychiatric services, either locally or elsewhere. About half had been referred *directly* to the shelters from the local psychiatric service. Of those who were technically 'off the streets', 85% had previously been in psychiatric hospitals, many of them for long admissions.

It is instructive to consider whether these data reflect a significant social change, as bed numbers reduce nationally. The homeless have always included some mentally ill in their ranks (Herzberg, 1985). An early anecdotal study of a common lodging house (London Council of Social Services, 1960) gives a graphic description of mentally ill 'inmates':

> "one man walked backwards and forwards, carrying a fish basket and talking aggressively and ceaselessly to himself.... Another man at a table nearby was speaking into an imaginary microphone, and jotting notes in an imaginary notebook.... Another packed and unpacked a little cardboard box with a great show of ritual. Anybody who had worked in a mental hospital could not fail to recognise some of the symptoms. The problem was why these men were in a lodging house at all."

Nonetheless, the writer estimated that only about 4% of the total were seriously mentally ill.

Crossley & Denmark (1969) surveyed male residents of a 60-bed Salvation Army hostel: 33% of these had previously been in a psychiatric hospital. Priest (1971) interviewed 79 residents in common lodging houses in Edinburgh; psychiatric morbidity was diagnosed in 75% of the residents, with 32% of the total diagnosed as schizophrenic. A study of the Camberwell Reception Centre by Tidmarsh & Wood (1972) found that, of new cases attending the centre, 22% suffered from mental illness, 14% had predominantly alcohol-related problems, and 16% were considered to have a personality disorder. Ten per cent of the sample had been admitted to a mental hospital in the previous year, and a further 19% more than a year previously. In a later paper, Wood (1976) reported that nearly two-thirds of those currently mentally ill at the centre were not receiving medical treatment, and that the centre was inappropriately acting as a home for the chronically mentally ill in the absence of suitable after-care facilities. These studies, unlike Marshall's, provided no information about the level of disability of those diagnosed as mentally ill.

In the United States, recent figures on the homeless mentally ill are alarming. Bassuk (1984) reports that 90% of residents of a Boston night shelter had a diagnosable mental disorder, while Baxter & Hopper (1984) report a number of studies that found psychiatric disorders present in 25–85% of homeless people.

The problem of psychiatric problems among the homeless is thus not new. However, it is quite possibly growing. In 1985 the House of Commons Social Services Committee accepted that there is an association between the rundown of mental hospital beds and the growth of the homeless population. Furthermore, it is likely that the *severity* of disability of psychiatric patients in the community is on the increase, although more studies will be needed which, like Marshall's, specifically gather these data.

The question is raised of how to provide a suitable psychiatric service. Should psychiatric hospitals be prevented from discharging their patients directly to such inadequate accommodation as shelters for the homeless? Or indeed, should those identified in these settings as severely disabled be returned to the hospital? Yet, at present, the health service is not recognised as the statutory agency for providing housing. Those admitted to psychiatric units often have accommodation problems. Ebringer & Christie Brown (1980) found that 28% of short-stay admissions to a London mental hospital had no or only temporary accommodation, and that during the course of the admission, 40% lost their accommodation. Thus, mentally-ill patients in hospital often require rehousing.

Apart from the problem of a shortage of suitable (often staffed) housing, the views of the service users are relevant. Given a lack of suitable permanent accommodation, do they prefer to remain in hospital wards, supervised by nurses, or in shelters, largely left to their own devices? Are they exercising a legitimate choice not to be treated, or being denied access to psychiatric care? Assuming that some will prefer not to remain on hospital wards, or that hospitals will continue to discharge them anyway, some responsibility for providing them with psychiatric services should be accepted. In a time of scarcity of resources, a high priority should be accorded to the provision by community psychiatric teams of services to shelters for the homeless, as an urgent social, psychiatric, and political need.

R. M. Toms

My children have become adept at spotting which television programmes Mum, as a rehabilitation psychiatrist, 'ought' to watch. More and more frequently, these deal with the closure of large psychiatric hospitals, and – with varying degrees of emotion and accuracy – the fate of their residents. Typically, the camera focuses on an imposing Gothic building surrounded by flower beds and rolling lawns, followed by a shot of 'Cardboard City' in one of our large towns, a crowded hostel, a lodging house in a dingy street, or a mouse-infested broken-down car abandoned by the roadside – all of which serve as 'home' for discharged patients. Rare indeed must be the old lady whose ward was demolished around her and who exchanged the echoing corridors of a large institution for an elegant Kensington mansion.

These scenes may be exaggerated, but Dr Marshall's work is firmly based on reality. He has carried out a painstaking and revealing study in two of these 'homes' – hostels for homeless people in Oxford.

His conclusions replicate the findings of other studies that homeless mentally-ill people tend to congregate in hostels originally intended as temporary shelters for travelling workers or vagrants. A survey carried out in Westminster to examine the whereabouts of patients discharged from short-stay psychiatric units over six months in 1988 found that one in four was homeless, and that in the capital's hostels, half of those seeking a bed for the night were mentally ill (Westminster Association for Mental Health, 1989). However, Dr Marshall's study goes further: he points out that previous studies, when considering residents of hostels for the homeless, have concentrated on demographic factors and diagnosis. His work is probably unique in that it aimed to assess the level of disability of these people by focusing on the intensity of their psychiatric symptoms and the degree of disturbance of their behaviour. Taken together, these aspects give a very accurate picture of the practical difficulties which hostel staff have to deal with day by day.

Trainees in psychiatry who come to a rehabilitation unit after a period in general acute psychiatry are often disconcerted to find that actual diagnosis frequently seems less important than behavioural abnormalities, social disabilities, and the ways in which a person's psychiatric symptoms affect behaviour. These three factors are crucial in determining potential for resettlement in the community: a former psychiatric patient should not be marked out as 'different'. An old man with well encapsulated delusions who will talk about them when encouraged but whose day-to-day behaviour seems unaffected will integrate more easily than his friend whose paranoid delusions make him suspicious of everyone he meets and who is occasionally abusive or violent as a result of these beliefs.

The industry and persistence shown in collecting data on such a difficult group of patients in this study is very impressive. Almost 50% of the residents had been referred by the Oxford psychiatric services, and only 10% of the total had had no psychiatric admissions. Mental state assessment showed that 70% of the sample had florid psychotic symptoms, and 79% symptoms of a deficit state. The behavioural rating scale revealed a high level of deviant behaviour (aggression, violence, sexual offensiveness, self-injury). General behaviour scores indicated that only 26% had abilities which, had the survey been used in a psychiatric hospital, would have indicated 'potential for discharge'. No fewer than 48% fell into the 'high-dependency' range, indicating very little ability to cope with daily living. In my own hospital, where REHAB rating scales were used some years ago to place all long-stay patients according to their level of functioning rather than diagnosis, people with high scores are either chronic psychotic patients who have been in hospital for many years and whose chances of resettlement in the community are slight, or else those who have a significant degree of physical illness in addition to their psychiatric problem and who require much nursing care. Many have behavioural disturbances which, although tolerated to a certain extent in the corridors of a large psychiatric hospital, would certainly shock, alarm, or annoy people in the average street or road.

Similar individuals to these, as Dr Marshall clearly shows, are being cared for in the two Oxford hostels – people who remain chronically and severely mentally ill and whose behaviour reflects this. His study raises several important questions, the answers to which must be found in further research.

Forty-eight per cent of the group studied had been referred to the hostels by Oxford psychiatric services. It is not clear how many were discharged there direct from hospital, possibly because of closure of beds, and how many had been living in other residential accommodation but were asked to leave there because of disturbed behaviour or florid symptoms. It seems that all but the 26% who had a 'potential for discharge' deserved a hospital bed. Were the psychiatric services satisfied that they were doing the best for their patients, or were they under pressure to discharge because of financial and policy reasons? Was alternative community accommodation available? How many of the 48% should have been admitted to hospital but refused treatment, and how many would have been admitted had a bed been available?

It would be interesting to know what level of follow-up was provided for patients referred by psychiatric services. The hostel staff and volunteers were not psychiatrically trained, and one wonders whether support and guidance was available to them from professional mental health workers. Were the staff prepared in any way for the problems they might have to face in dealing with this group of people? Untrained staff can often become demoralised and despairing if they feel unsupported and unappreciated, especially when faced with unfamiliar and sometimes frightening aspects of behaviour.

"We are witnessing the inadvertent creation of mini institutions in hostels" says Dr Marshall. The idea of "mini institutions" is currently unpopular, as they imply little effort to promote rehabilitation techniques, but I believe that they are not necessarily bad for a certain group of people who would feel confined and disorientated in a small house but who could take advantage of some move towards domesticity and 'normalisation'. Dr Marshall hints that the hostels are able to manage their very difficult task because of the devotion and ingenuity of the staff who had adopted 'impressive strategies' to deal with the challenge. Support, continuing training, and follow-up of residents by professionals could make their work even more effective and less of a burden. In north-east Essex, a large number of former psychiatric patients live in former seaside boarding houses in Clacton, and a specialised community rehabilitation team has been recently established to care for people with long-term psychiatric problems living in the town. Members of this team are frequent visitors to individual boarding houses to offer advice on management of specific clinical problems, and a proprietors' support group has recently been formed.

The need for a whole range of community accommodation for all levels of disability will become increasingly necessary as long-stay hospitals close. We must be sure that the best possible care continues to be given to a much neglected group of people. It is up to mental health professionals, particularly those in the rehabilitation field, to ensure that their skills are used not only to treat patients with long-term needs directly, but to support, teach, and encourage others to work effectively alongside them.

Properly organised and supervised, community care can be very successful, but we must ensure that all aspects of a community service receive maximum help and encouragement to meet the clients' needs.

References

BASSUK, E. L. (1984) The homelessness problem. *Scientific American*, **251**, 28–33.

BAXTER, E. & HOPPER, K. (1984) Troubled on the streets: the mentally disabled homeless poor. In *The Chronic Mental Patient: Five Years Later* (ed. J. A. Talbott). Florida: Grune & Stratton.

CROSSLEY, B. & DENMARK, T. C. (1969) Community care: a study of the psychiatric morbidity of a Salvation Army hostel. *British Journal of Sociology*, **20**, 443–449.

EBRINGER, E. & CHRISTIE BROWN, J. R. W. (1980) Social deprivation among short-stay psychiatric patients. *British Journal of Psychiatry*, **131**, 46–52.

HERZBERG, J. L. (1985) No fixed abode: a comparison of male and female admissions to an East London psychiatric hospital. Unpublished MPhil thesis, University of London.

LONDON COUNCIL OF SOCIAL SERVICES (1960) *Forgotten Men*. London: NCSS.

MARSHALL, M. (1989) Collected and neglected: are Oxford hostels for the homeless filling up with disabled psychiatric patients? *British Medical Journal*, **299**, 706–709.

PRIEST, R. G. (1971) The Edinburgh homeless. *American Journal of Psychotherapy*, **25**, 194–213.

TIDMARSH, D. & WOOD, S. (1972) Psychiatric aspects of destitution. In *Evaluating a Community Psychiatric Service* (eds J. K. Wing & A. M. Hailey). Oxford: Oxford University Press.

WESTMINSTER ASSOCIATION FOR MENTAL HEALTH (1989) *Is Community Care Working?* London: WAMH.

WOOD, S. M. (1976) Camberwell Reception Centre. *Journal of Social Policy*, **5**,389–399.

Philippa Garety, MA, MPhil, AFBPsS, *Lecturer in Psychology, Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF*; Rhinedd M. Toms, MA, MB, BChir, MRCPsych, *Consultant Psychiatrist, Mental Health Unit, Severalls Hospital, Boxted Road, Colchester, Essex CO4 5HG*

British Journal of Psychiatry (1990), 157, 273–275

Brief Reports

Is Parental Age Related to the Risk of Alzheimer's Disease?

ALBERT HOFMAN, CORNELIA M. van DUIJN, WILLEM SCHULTE, THEUNIS A. TANJA, ROBERT HAAXMA, ARIE J. LAMERIS and ROLF J. SAAN

Advanced maternal and paternal age were investigated as putative risk factors for AD in 198 clinically diagnosed Alzheimer patients and in 198 randomly selected healthy controls. No significant differences in average age of fathers and of mothers at birth of the subject were observed. The risk of AD was not significantly different across categories of maternal and paternal age. The association with parental age was not different for sporadic and familial AD. These findings do not support the view of a maternal or paternal age effect on AD.

Advanced parental age has been suggested as a putative risk factor for Alzheimer's disease (AD) because of its clinical and pathogenetic links with Down's syndrome. However, studies of this subject have yielded equivocal results. In four studies the offspring of old mothers had a higher risk of developing the disease than those of young mothers (e.g. Cohen *et al*, 1982; Amaducci *et al*, 1986) but this was not the case in eight others (e.g. De Braekeleer *et al*, 1988; Jouan-Flahault *et al*, 1989). In the largest study, with 116 clinically diagnosed Alzheimer's patients and 97 population controls, maternal age over 40 years was associated with a nearly five-times increased risk of AD (Amaducci *et al*, 1986). A recent additional analysis of that study showed that the increase in risk was restricted to sporadic Alzheimer patients (those with no family history of AD); in cases with familial AD the relative risk was not elevated (Schoenberg *et al*, 1988). We report a study of the putative association of AD and parental age in 198 Dutch Alzheimer patients who were compared with 198 population controls.

Method

The ages of the mother and father at birth of 198 Alzheimer patients and 198 age- and sex-matched healthy population controls were assessed, as part of a case-control study of Alzheimer's disease in the Netherlands. This study has been described in detail elsewhere (Hofman *et al*, 1989). In the patients the diagnosis of AD was made before the age of 70 years.

Patients

All patients in whom the clinical diagnosis of AD was made in the period of January 1980 to July 1987, who were not yet 70 years at the time of diagnosis, and who lived in the four northern provinces of the Netherlands or in the region of the city of Rotterdam, were eligible for this study. All patients in these areas were seen by three of the authors (WS, TAT and RH). Dementias other than AD were excluded using the clinical history, a neurological examination, and neuropsychological and laboratory tests (McKhann *et al*, 1984). The diagnosis of AD was made in patients fulfilling the following criteria: (a) slow progressive decline of intellectual function; (b) Clinical Dementia Rating scale score of >0.5 (Hughes *et al*, 1982); (c) Short Portable Mental Status Questionnaire (SPMSQ) score of <20 (Pfeiffer, 1975); (d) Hachinski scale score of ⩽7 (Hachinski *et al*, 1974); and (e) no evidence for abnormalities other than cerebral atrophy, on a computerised tomography (CT) and no evidence for focal dysfunction on an electroencephalograph (EEG). Of the 278 patients brought to our attention, 201 satisfied these criteria, and in 198, data on putative risk factors for AD were obtained.

Controls

For each patient a reference subject was randomly selected from the municipal population register, matched for age (within 5-year age-groups) and gender, and living in the same municipality as the patient at the time of diagnosis. All controls were apparently healthy, with a normal cognitive function and an SPMSQ score of ⩾20.

Data collection and analysis

The age of the parents at birth of the Alzheimer patient and the birth order of the patient and his/her siblings were obtained from the next of kin (in general the wife or husband, or a child), as part of a structured interview on risk factors for AD. The parental age in the control subjects was also obtained in the same way. Reliable information on maternal age and birth order was obtained in 184 patients and controls, and on paternal age in 160 patients and controls. The data were checked either by using the municipal population register or information from a second family member. Full information was collected on dementia in the family. Only those with at least one first-degree relative with dementia were considered to have a familial AD.

The strength of the association of maternal and paternal age and AD was assessed by computation of the relative risk (odds ratio), with 'parents younger than 25 years at birth of the patient or control' as the reference category. The analysis was done for all cases and then separately

TABLE I

Age of parents at subject's birth and relative risk for AD in all patients and those with sporadic AD

Age of parent at subject's birth: years	*Mothers*				*Fathers*			
	No. of patients	*No. of controls*	*RR*[1]	*95% CI*[3]	*No. of patients*	*No. of controls*	*RR*[1]	*95% CI*[3]
All patients								
≤24	52	46	1.0[2]		27	21	1.0[2]	
25–29	54	52	0.9	0.5–1.6	50	42	0.9	0.5–1.9
30–34	45	45	0.9	0.5–1.6	43	45	0.7	0.4–1.5
35–39	18	29	0.6	0.3–1.1	18	32	0.4	0.2–1.0
40+	15	12	1.1	0.5–2.6	22	20	0.9	0.4–1.8
Total	184	184			160	160		
Patients with sporadic AD								
≤24	28	24	1.0[2]		13	12	1.0[2]	
25–29	25	27	0.8	0.4–1.7	25	22	1.1	0.4–2.8
30–34	24	24	0.9	0.4–1.9	21	20	1.0	0.4–2.6
35–39	10	12	0.7	0.3–1.9	10	13	0.7	0.2–2.2
40+	7	7	0.9	0.3–2.8	9	11	0.8	0.2–2.5
Total	94	94			78	78		

1. Relative risk (RR) adjusted for age, sex and area of residence.
2. Reference category.
3. 95% confidence intervals.

TABLE II

Differences in mean (standard error of mean) age of parents at birth of patients and controls by family history of dementia

Family history of dementia	*Mean mothers' age: years*				*Mean fathers' age: years*			
	patients (s.e.m)	*controls (s.e.m)*	*difference (s.e.m)*	*95% CI*	*patients (s.e.m)*	*controls (s.e.m)*	*difference (s.e.m)*	*95% CI*
Yes	28.4 (0.7)	29.6 (0.6)	1.2 (0.9)	−0.6, 3.0	31.1 (0.9)	32.5 (0.8)	1.4 (1.2)	−1.0, 3.6
No	28.8 (0.7)	29.1 (0.6)	0.3 (0.9)	−1.5, 2.1	30.4 (0.7)	32.0 (0.8)	1.6 (1.1)	−0.6, 3.8
Total	28.6 (0.5)	29.3 (0.5)	0.7 (0.7)	−0.7, 2.1	30.7 (0.5)	32.2 (0.6)	1.5 (0.8)	−0.1, 3.1

for sporadic cases (no first-degree relatives with Alzheimer's disease) only. The relative risk is presented with a 95% confidence interval (95% CI). The matching variables (age, gender and area of residence) were taken into account by entering them into a model for logistic regression (Schlesselman, 1982). The reported relative risks are based on the regression coefficient yielded by this model. A separate matched-pair case-control analysis showed similar results. In an additional analysis, the mean age of the mother and father at birth of the subject was compared for patients and controls.

Results

The risk of AD was not significantly different across categories of maternal and paternal age at birth of the subject (Table I). The relative risk for maternal age of 40 years or over was 1.1 (95% CI 0.5–2.6). In sporadic (non-familial) Alzheimer patients the risk was also not different over strata of parental age (Table I). The relative risk for maternal age of 40 years or over was 0.9 (0.3–2.8). No significant differences in average ages for mothers and fathers at birth of the subject were observed (Table II). A separate analysis showed no significant differences in birth order between Alzheimer patients and controls. This applied both to those with and those without a family history of AD.

Discussion

There is much evidence to suggest that genetic factors play a part in the aetiology of AD. In epidemiological studies, strong familial aggregation of Alzheimer patients has been observed, and recently, genetic linkage between AD and marker loci on chromosome-21 has been reported (St George-Hyslop *et al*, 1987). There is also evidence for clinical and neuropathological links between AD and Down's syndrome (Anonymous, 1987). These observations have led to the hypothesis that advanced parental age may cause abnormalities of chromosome-21, which may lead to AD.

This study fails to confirm previous findings of a maternal age effect on the risk of AD. The average age of the parents at birth of the subjects was, if anything, slightly lower in the patients than in the controls. This study differs from others in that it is considerably larger, and employed randomly selected population controls.

Two methodological issues have to be discussed. Firstly to reduce misclassification of Alzheimer patients as much as possible we applied the diagnostic criteria suggested by McKhann *et al* (1984) rigorously and restricted our study to patients in whom the diagnosis was made before the age of 70 years. However, we cannot rule out that some misclassification has occurred and as in other studies this tends to dilute a true difference in maternal age between the study groups. Secondly, this study was performed in early-onset Alzheimer patients. We do not know to what extent the present findings apply to AD with a later onset.

In conclusion, we suggest that parental age at birth of the subject is not likely to be a risk factor for AD.

Acknowledgement

The authors thank Verona Otten for her contribution to data-collection, and Drs H. A. Valkenburg and H. W ter Haar for their advice. This study was supported by the SOOM foundation; it is part of the Eurodem EC Concerted Action on the Epidemiology of Dementia

References

Amaducci, L. A., Fratiglioni, L., Rocca, W. A., *et al* (1986) Risk factors for clinically diagnosed Alzheimer's disease: a case-control study of an Italian population. *Neurology*, **36**, 922–931.

Anonymous (1987) Alzheimer's disease, Down's syndrome, and chromosome 21. *Lancet*, *i*, 1011–1012.

Cohen, D., Eisdorfer, C. & Leverenz, J. (1982) Alzheimer's disease and maternal age. *Journal of the American Geriatric Society*, **30**, 656–659.

De Braekeleer, M., Froda, S., Gantrin, D., *et al* (1988) Parental age and birth order in Alzheimer's disease: a case-control study in the Saguenay-Lac-St-Jean area (Quebec, Canada). *Canadian Journal of Neurology and Science*, **15**, 139–141.

Hachinski, V. C., Lassen, N. A. & Marshell, J. (1974) Multi-infarct dementia: a cause of mental deterioration in the elderly. *Lancet*, *ii*, 207–209.

Hofman, A., Schulte, W., Tanja, T. A., *et al* (1989) History of dementia and Parkinson's disease in first-degree relatives of patients with Alzheimer's disease. *Neurology*, **39**, 1589–1592

Hughes, C. P., Berg, L., Domziger, W. L., *et al* (1982) A new clinical scale for the staging of dementia. *British Journal of Psychiatry*, **140**, 566–572.

Jouan-Flahault, C., Seroussi, M. C. & Colvez, A. (1989) Absence de liaison entre démence sénile et âge parental *Revue d'Epidémiologie et de Santé Publique*, **37**, 73–76

McKhann, G., Drachman, D., Folstein, M., *et al* (1984) Clinical diagnosis of Alzheimer's disease: report of a NINCDS-ADRDA Work Group. *Neurology*, **34**, 939–944.

Pfeiffer, E (1975) A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. *Journal of the American Geriatric Society*, **23**, 433–441.

Schlesselman, J. J. (1982) *Case-Control Studies*. New York: Oxford University Press.

Schoenberg, B. S., Rocca, W. A., Fratiglioni, L., *et al* (1988) Late maternal age as a risk factor for sporadic and familial Alzheimer's disease. *Neurology*, **38**, 311.

St George-Hyslop, P. H., Tanzi, R., Polinsky, R. J., *et al* (1987) The genetic defect causing familial Alzheimer's disease maps on chromosome 21. *Science*, **235**, 885–890.

*A. Hofman, MD, PhD, *Professor of Epidemiology, Department of Epidemiology and Biostatistics, Erasmus University Medical School, PO Box 1738, 3000 DR Rotterdam, The Netherlands*; Cornelia M. van Duijn, MSc, *Resident in Epidemiology*; Willem Schulte, MD, *Social Geriatrician*; Teun A. Tanja, MD, *General Practitioner*; Robert Haaxma, MD, PhD, *Lecturer in Clinical Neurology*; Arie J. Lameris, MD, *General Practitioner*; Rolf J. Saan, MSc, *Reader in Neuropsychology*

*Correspondence

British Journal of Psychiatry (1990), **157**, 275–278

The Effects of Chronic Lithium Treatment on Psychomotor Performance Related to Driving

SIMON HATCHER, RUTH SIMS and DAVID THOMPSON

A group of 16 psychiatric out-patients in remission, who had been taking lithium carbonate as their sole medication for at least three months, were compared with a control group of

22 healthy volunteers. On a computerised driving simulator which produced measures of reaction time, tracking ability and mistakes made, the patient group had a significantly slower reaction time. Patients shou'd be warned therefore that lithium may affect their ability to drive or operate machinery and that psychomotor impairment and sedation are not synonymous.

Medical Aspects of Fitness to Drive (Raffle, 1985) published by the Medical Commission on Accident Prevention, states that patients "should be advised of the effects of [prophylactic] medication" in relation to driving ability. Previous work on relating lithium to driving skills has involved either normal volunteers given medication for a short period (Linnoila *et al*, 1974) or small numbers of patients taking other medication concurrently (Hobi *et al*, 1982). These studies have come to different conclusions concerning the safety of driving while taking lithium.

The validity of using laboratory tests to relate drug effects to driving skills has been demonstrated by prospective follow-up studies (Haakinen, 1976) and by comparing laboratory tests with other tests of driving skills (Sivak *et al*, 1981). This study aimed to examine the effects of chronic treatment with lithium carbonate on the driving skills of psychiatric out-patients in remission, using a computerised driving simulator.

Method

Patients were entered into the study if they were out-patients, were taking lithium as their sole psychotropic medication, had been on lithium for at least three months, and were between the ages of 18 and 65 years. They were excluded if they were actively psychiatrically ill or had any concurrent neurological disease. In practice, all the patients included in the study had previously been diagnosed as suffering from manic–depressive psychosis – circular type.

The controls were volunteers recruited from health authority domestic, secretarial, medical and nursing staff who were not on any medication and did not have any concurrent physical or psychiatric illness. Their occupations were as follows: four doctors; eight psychiatric nurses; four secretaries; two medical students; one domestic; one social worker; one salesman; and one research assistant. They were matched for age, sex, and driving experience with the patient population. All the drivers in the control and patient group were currently driving.

The null hypothesis was that there was no difference in psychomotor abilities between the two populations. All investigations, with the exception of those two patients who were tested in the afternoon, were performed in the morning in order to control for circadian variation in reaction time.

Psychomotor ability was assessed using a BBC microcomputer and a simple driving simulator. This consisted of a steering wheel and two foot pedals positioned to replicate the brake and accelerator pedals in a car. The screen display consisted of a roadway with two traffic lights either side of the road and two arrows at the bottom of the screen. The traffic lights changed colour at random time intervals 11 times a minute. The top arrow was moved by the computer in a horizontal plane according to a complex sine wave and the bottom arrow by the subject using the steering wheel. Subjects were instructed to follow the computers' arrow using the steering wheel and at the same time, if either of the traffic lights changed from green to red, to take their foot off the accelerator pedal and press the brake pedal as fast as possible. This generated an accuracy or tracking score, which reflected how closely the subjects followed the computers' arrow, a brake reaction time and a record of how many lights the subject had failed to respond to (lights missed). The closer the match between the computers' arrow movement and that of the subject the lower the accuracy score, i.e. low scores represented higher accuracy (the score was derived from the inverse of a root mean square measure of the deviation of the subject's arrow from the computer-generated track).

Subjects were given a five-minute practice period on the simulator during which 55 changes of the lights were made. They were then tested for three minutes when there were 33 changes of the lights.

To confirm that they were in remission, patients also completed a Hospital Anxiety and Depression Rating Scale (HAD scale) and were interviewed using the Montgomery-Asberg Depression Rating Scale (MADRAS scale). Patients were excluded if they scored seven or more on the MADRAS scale, which corresponds to a 'mild depressive disorder' (Snaith *et al*, 1986), and eight or more on the HAD depression subscale, which indicates a probable case of depression (Zigmond & Snaith, 1983). Blood samples were collected for serum lithium measurement, thyroid function tests and blood alcohol. Patients were excluded if their thyroxine levels were outside the normal range of 60–140 nmol/l or if there was any alcohol in their blood.

Results

A comparison of some of the demographic features of the two groups is shown in Table I. The patients were taking a mean dose of lithium carbonate of 809 mg daily (range of 400–1250 mg daily), and had been on this medication for a mean of 4.36 years. At the time of taking the test the mean serum lithium was 0.61 mmol/l (range 0.27–1.04 mmol/l). Two patients were included with serum lithium levels below the usually accepted minimum of 0.40 mmol/l who had been successfully maintained on a low dose of lithium for some years.

A comparison of the two groups on the driving simulator is shown in Table II. This shows that the patients had a significantly slower reaction time than controls. The difference in accuracy approached significance while there was no significant difference in the number of mistakes (lights missed). However, because of the numbers involved, the power of the statistical tests to detect a significant difference is probably low.

TABLE I
Composition of control and patient groups

	Controls (n = 22)	*Patients* (n = 16)
Age range: years	21–58	18–63
Mean age: years	37.8	36.0
Females/males	12/10	8/8
Non-drivers (%)	5 (23)	4 (25)

TABLE II
Comparison of control and patient groups on the driving simulator (all statistical tests are two-tailed Mann–Whitney U-tests)

	Controls	*Patients*
Median reaction time: seconds (range)	0.58 (0.46–0.82)	0.72* (0.50–1.01)
Median accuracy[1] (range)	27.5 (16–51)	32** (24–75)
Median lights missed: *n* (range)	0.00 (0–8)	0.00*** (0–4)

*$P<0\,05$, **$P=0.052$, ***$P=0.23$.

1. Score derived from inverse of a root mean square measure of the deviation of subjects' arrow from computer-generated track.

Within the patient group there was no significant correlation between age and accuracy (Spearman's $r=-0.08$, $P=0.75$), reaction time ($r=0.30$, $P=0.27$) or lights missed ($r=0.30$, $P=0.27$) or between anxiety scores from the HAD and reaction times ($r=0.18$, $P=0.53$). Serum lithium and total thyroxine (T_4) level were not significantly correlated with any of the variables measured on the driving simulator. In an attempt to further examine the effects of the serum lithium level the patients were divided by means of the median lithium level into a high and a low group. There were no significant differences between either group on any of the variables. Excluding non-drivers from the analysis confirmed a significant difference in reaction time ($P<0.05$) with the difference in accuracy approaching significance ($P=0.53$).

Discussion

Lithium carbonate is a commonly used drug in psychiatric out-patients and yet, like most drugs, its effects on everyday activities such as driving have received little attention. This study has shown that compared to a normal population, patients taking lithium have an increased reaction time. Is this a true difference?

It could be argued that lithium slows down the learning process. Therefore, what this study measures is reaction times at different points on the same learning curve and, given sufficient practice, both groups would eventually reach the same level. However, the tasks on the simulator were easy to learn and after the initial instructions, all the subjects could be left to operate the simulator on their own. It has also been shown that reaction time measurements are stable and reproducible after the first 18 trials in a series (Hamsher & Benton, 1977). Subjects in this study had 55 trials before measurements were taken.

Is the difference in reaction time due to the underlying disease process or an effect of the drug? Lithium does have specific psychomotor effects (Shaw *et al*, 1987). Elsass *et al* (1981) found a significant impairment in reaction times comparing lithium-treated patients who had 'manic-melancholic' disorder with patients who had the same diagnosis but were not being treated with lithium. However, these results have to be treated with caution because no allowance was made for the affective state of the subjects or any other medication that they might have been taking. In the present study a better experimental design would have been to use the patients as their own controls (i.e. take measurements both on and off lithium). However, this has obvious ethical problems. Whether the difference was due to the disease or the drug, the important point is that it exists. What is also important is that a drug which does not cause sedation at therapeutic levels does have an effect on psychomotor abilities. Sedation and psychomotor impairment are not synonymous.

Does a difference of 0.14 s really matter? Driving at 30 m.p.h. the 'thinking distance' is 9.1 m according to the Highway Code. An increase in thinking time of 0.14 s results in an increase of about 2 m or a 22% rise. It may be argued that patients adopt alternative strategies to cope with this change and are therefore perfectly safe. However, first they must be aware of the change and it is the prescribing doctor's duty to warn patients given lithium that their ability to drive may be affected.

Acknowledgements

We are grateful to Dr T. Jerram for help in providing patients and to Mr P. Preston and Dr H. Wildgust of Dista Products Ltd for supplying the driving simulator.

References

ELSASS, P., MELLERUP, E. T., RAFAELSEN, O. J., *et al* (1981) Effect of lithium on reaction time – a study of diurnal variations. *Psychopharmacology*, 72, 279–282.

HAAKINEN, S. (1976) Traffic accidents and psychomotor test performance. A follow-up study. *Modern Problems in Pharmacopsychiatry*, 11, 51–56.

HAMSHER, K. S. & BENTON, A. L. (1977) The reliability of reaction time determinants. *Cortex*, **13**, 306–310.

HOBI, V., GASTPAR, M., GASTPAR, G., *et al* (1982) Driving ability of depressive patients under antidepressants. *Journal of International Medical Research*, **10**, 65–81.

LINOILA, M., SAARIO, I. & MAKI, M. (1974) Effect of treatment with diazepam or lithium and alcohol on psychomotor skills related to driving. *European Journal of Clinical Pharmacology*, **7**, 337–342.

RAFFLE, A. (ed.) (1985) *Medical Aspects of Fitness to Drive.* London: The Medical Commission on Accident Prevention.

SIVAK, M., OLSON, P. L., KEWMAN, D G., *et al* (1981) Driving and perceptual/cognitive skills: behavioural consequences of brain damage. *Archives of Physical Medicine and Rehabilitation*, **62**, 476–483.

SHAW, E. D., STOKES, P. E., MANN, J. J., *et al* (1987) Effects of lithium carbonate on the memory and motor speed of bipolar out-patients. *Journal of Abnormal Psychology*, **96**, 64–69.

SNAITH, R. P., HARROP, F. M., NEWBY, D. A., *et al* (1986) Grade scores of the Montgomery-Asberg Depression and Clinical Anxiety Scales. *British Journal of Psychiatry*, **148**, 599–601.

ZIGMOND, A. S. & SNAITH, R P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

*Simon Hatcher BSc, MBBS, *Tutor in Psychiatry and Honorary Registrar in Psychiatry, Department of Psychiatry, University of Leeds, Leeds*; Ruth Sims, MBChir, MRCPsych, *Tutor in Psychiatry and Honorary Registrar in Psychiatry, High Royds Hospital, Menston, Ilkley*; David Thompson MBBS, MRCPsych, MSc, *Consultant Psychiatrist, Malham House Day Hospital, Leeds*

*Correspondence: *Department of Liaison Psychiatry, Leeds General Infirmary, Great George Street, Leeds LS1 3EX*

British Journal of Psychiatry (1990), **157**, 278–281

Do-Do Abuse

S. LOOSMORE and D. ARMSTRONG

Three cases of prolonged abuse of Do-Do tablets, an over-the-counter remedy for "coughs, wheezing and breathlessness", are reported. They have an amphetamine-like action and were used as easily obtained amphetamine substitutes, in one case to relieve social anxiety. Withdrawal symptoms similar to those following cessation of amphetamines occurred in two cases. Do-Do tablets are CNS stimulants and their abuse may be accounted for by the fact that they perhaps affect amine neurotransmitters.

Many over-the-counter remedies for coughs and colds contain one or more central nervous system (CNS) stimulants. Abuse of such medication is recognised, but, until recently, infrequently reported in the medical literature. Most previous reports have concentrated on the acute sequelae of long-term use or abuse.

Do-Do tablets, containing ephedrine hydrochloride (222 mg), caffeine (30 mg), and theophylline sodium glycinate (50 mg), are widely available over the counter and marketed for the relief of "bronchial coughs, wheezing and breathlessness". The packet instructions are brief, stating that no more than three tablets should be taken in twenty-four hours. There is no comment on possible side-effects or the consequences of exceeding the stated dose.

We report, in detail, three cases of prolonged abuse of Do-Do tablets.

Case 1

A 33-year-old, separated, unemployed man was referred to the addiction services with a 15-year history of abuse of Do-Do tablets. He feared that his drug use would prevent access to his children. His stated aim was to abstain.

His mother and maternal grandfather have suffered endogenous depression. His father and paternal uncle are withdrawn, inaccessible individuals and his paternal grandfather, although not formally diagnosed, was an odd man.

Since childhood he had been reclusive with difficulty in forming stable relationships. At school he would sit curled up in a corner with gaze averted, and without speaking. Because of his evident oddness he was bullied.

As a child he was diagnosed as asthmatic. At eighteen he discovered that Do-Do tablets had the dual benefits of relieving his dyspnoea and producing feelings of euphoria. He rapidly increased his consumption to an average of 30 tablets daily; this dose continued unchanged until his referral. In addition to Do-Do tablets he had, in the past, intermittently taken up to one gram of oral amphetamine sulphate daily although his preference was always for Do-Dos. His alcohol consumption was regular but moderate, averaging nine pints of cider weekly.

In 1984 he had a brief admission to a psychiatric hospital because of persecutory ideation. He had been carrying a knife for protection. He did not reveal his use of Do-Do tablets and the symptoms were felt to be due to amphetamine abuse. On other occasions, having taken large amounts of amphetamine, he would experience transient suspiciousness. He had stopped using amphetamine without difficulty three years before referral.

He had a stormy, short-lived marriage to an emotionally unstable ex-prostitute. After their separation, two years before referral, he became miserable, increasingly withdrawn and morbidly ruminative. Apart from Do-Do hunting he rarely ventured out.

He was admitted for assessment. He was emotionally flat with poor rapport and eye contact. He was vague and preoccupied with his marital breakdown. There was no thought disorder, nor any abnormal ideas or perceptions. Cognitive testing was normal as was physical examination. There was little somatic disturbance and he was not clinically depressed. Routine laboratory tests were normal and HIV and syphilis serology were negative.

The Do-Dos were stopped abruptly. Over the next forty-eight hours he became listless, muddled and had difficulty concentrating. He developed ideas of reference, believing that fellow patients were discussing him. Within ten days these symptoms had resolved without medication. Spontaneity, eye contact and mood all improved. He began to complain of intermittent chest tightness without evidence of bronchospasm. Within a few days of discharge he resumed Do-Do use in similar quantities as before admission.

Case 2

A 46-year-old, separated, unemployed man living with his parents is seen regularly as an out-patient, as he receives maintenance methadone (25 mg daily), and diazepam (25 gm daily). He drinks a litre of Crème de Menthe a week. He has been taking 9–12 Do-Do tablets daily for 30 years although his consumption decreased when also using amphetamines. He believes that Do-Do tablets have no psychological effects after such prolonged usage, but that were he to stop he would become depressed: "they help me to cope, warding off depression." He has no intention of stopping or reducing his consumption.

He obtains his supplies from a local chemist who informally regulates the number he consumes by claiming occasionally that he has run out of stock.

Since the age of 19 he has abused a wide variety of psychoactive drugs including cannabis, barbiturates, codeine, heroin, amphetamine, cocaine and cyclizine and considers that Do-Dos closely resemble amphetamine in their effects. He has, in the past, injected using shared equipment but no longer has venous access.

He has no psychiatric history apart from drug abuse. His father suffered a depressive illness in the past and is now in the early stages of dementia. He has not worked for several years, but in the past has had several jobs including psychiatric nurse, civil engineer and factory shift manager.

Mental state examination shows an intelligent, erudite, well-spoken man. His talk is vague and circumstantial and he occasionally drifts from the point. His humour is self-mocking and he establishes fair rapport. His mood is neutral and there is no evidence of cognitive impairment.

Case 3

A 36-year-old divorced lady is seen in the Addiction Unit out-patients clinic in order to regulate her intake of benzodiazepines and administer her twice-monthly fluphenazine injection. She is said to suffer from chronic schizophrenia complicated by poly-drug abuse.

Her upbringing was chaotic. Her mother drank heavily and her father was physically violent. There is a strong family history of drug and alcohol abuse. Her current cohabitee has a longstanding alcohol problem, abuses amphetamines and is physically violent towards her.

She first became psychiatrically ill aged 16 years since when she has had numerous hospital admissions with acute psychotic episodes characterised by disturbed behaviour, thought disorder and third-person auditory hallucinations. When she complies with depot medication it is effective in controlling acute relapses. She has an extensive history of overdoses, wrist slashing and manipulative behaviour. There is a long history of minor offending, including soliciting, and she has spent several short periods on remand.

She started experimenting with drugs aged 15 and over the years has abused a wide variety of compounds including alcohol, chloral hydrate, benzodiazepines, barbiturates, amphetamines, cocaine, heroin, LSD and solvents. Her stated preference is for benzodiazepines and stimulants. She uses both orally and intravenously and at times will take combinations of whatever drugs are available.

She began using Do-Do tablets 15 years ago after being told of their stimulant effects by a fellow psychiatric patient. She regularly takes 15–30 tablets a day, purchasing them from a variety of chemists. She says that they help keep her awake, give her more energy and increase her sense of well-being. Although she prefers the effect of amphetamine sulphate, Do-Do tablets are cheaper and easier to obtain. She states that when she has suddenly stopped using Do-Dos she has become depressed, lethargic and slept excessively for periods of up to seven days. She has intermittently abused a number of other over-the-counter preparations containing sympathomimetics, but never for longer than a few weeks at a time. She does not feel that her abuse of Do-Dos or other drugs is related to her psychotic breakdowns.

She presents as a bizarrely dressed woman who attempts but fails to look younger than her years. She makes poor eye contact but answers spontaneously. Her speech is vague, circumstantial and impoverished. Her thought processes tend to be concrete. When well she has good insight into her abnormal experiences.

Discussion

Drugs with a central stimulant action (e.g. sympathomimetic amines and methylxathines) are frequent constituents of over-the-counter preparations which are widely available through retail pharmacists. Until recently little has been written about the abuse potential of these preparations. The first report of ephedrine abuse appeared in the English literature 20 years ago (Herridge & A'Brook, 1968). This and subsequent reports have concentrated on the acute psychosis induced by prolonged or excessive use (e.g. Lambert, 1987; Whitehouse & Duncan, 1987). Long-term patterns of abuse are less well documented. Pugh & Howie (1986) described a patient who abused Actifed (containing pseudoephedrine and triprolidine) for its stimulant properties. Jelley (1987) briefly reported the abuse of Phensedyl (containing ephedrine, codeine phosphate and promethazine) among young men in a small country town. Whitehouse & Duncan (1987) reviewed 20 case reports of ephedrine psychosis. Of these 20 cases, three patients took ephedrine purely for its stimulant properties, two were asthmatic but increased the dose of ephedrine for its stimulant properties and two others were poly-drug abusers. The authors stated that, in the majority of the 20 cases, the dose of ephedrine gradually increased with length of use.

All the constituents of Do-Do tablets are CNS stimulants. Moderate doses of caffeine (100–300 mg, equivalent to 3–10 Do-Do tablets or 1–4 cups of instant coffee) may improve some aspects of psychomotor and intellectual performance (Rall, 1985). Larger doses (>300 mg) can alter mood (Lader & Bruce, 1986), although studies have produced contradictory findings on the magnitude and direction of the effect (Gilbert, 1988). In human behavioural studies, caffeine has an inconsistent reinforcing effect producing an increase in subjective drug-liking. This effect has marked individual variation and may be potentiated by physical dependence on caffeine (Griffiths & Woodson, 1988).

Theophylline sodium glycinate contains 51% anhydrous theophylline. Compared to caffeine it has a more powerful stimulant action on the CNS. At doses just above the therapeutic range it causes nervousness, tremor, insomnia and restlessness. Higher doses can cause convulsions. The stimulant effects are not well studied as they are principally seen as side-effects of asthma (Rall, 1985).

Ephedrine is structurally similar to amphetamine although its stimulant effect is said to be considerably less. The central effect of both is due to the release of naturally occurring amines from pre-synaptic nerve terminals in the CNS. Caffeine and theophylline are methylxanthines; the majority of their actions at therapeutic doses are due to antagonism of the inhibitory effect of adenosine on sympathetically released noradrenalin (Williams & Jarvis, 1988). It is therefore likely that all the constituents of Do-Do tablets affect amine neurotransmitters, and this could account for the abuse potential of this particular combination.

As shown by the case reports, Do-Do tablets have a subjective amphetamine-like action and in all three cases described were used as a substitute for amphetamine. The first patient used them as self-medication to relieve social anxiety and may have experienced a drug-induced dysphoric state which resolved with drug withdrawal. In this case physical symptoms following withdrawal were mild although he did experience brief paranoid ideation. The second and third patients reported abused Do-Dos as an easy substitute for amphetamines. Two of the cases experienced withdrawal symptoms similar to those following cessation of amphetamine. In all cases the dosage consumed varied little over long periods. All three cases can be said to show mild dependence in that they showed, to varying degrees, a compulsion to take the drug, stereotyped drug-taking behaviour, and salience of drug use. One case exhibited a desire to stop the drug followed by reinstatement of excessive use after a period of abstinence.

In the USA, abuse of stimulant-containing medicines has been recognised as a major problem (Doughty, 1982; Lake & Quirk, 1984). Ephedrine has been marketed as a cocaine substitute (Siegal, 1980) and has been a constituent of 'look-alike' tablets manufactured to resemble amphetamine in appearance and effect. These 'look-alikes' or 'pseudo-speed' tablets contained various combinations of proprietary drugs such as phenylpropanolamine, ephedrine and caffeine. In the early 1980s they were reported to be the third most common drug of abuse among New York adolescents, and their manufacture to be a multi-million dollar business (Dougherty, 1982). Concern over the abuse of proprietary stimulants led the Food and Drugs Administration to introduce legislation restricting the manufacture of certain combinations of stimulant drugs (Lake & Quirk, 1984).

In the UK there are many over-the-counter preparations containing one or more compounds with stimulant properties. The extent to which these preparations are abused is uncertain. It is a problem recognised by pharmacists (Anonymous, 1987; Harrison, 1988), and anecdotal evidence suggests that it is not uncommon among illicit drug users.

While these preparations remain easily available they are likely to be abused, and not only by patients attending addiction services. Information about over-the-counter drug use should form part of every psychiatric history.

References

Anonymous (1987) Drug abuse. *Pharmaceutical Journal*, **239**, 117.

Dougherty, R. J. (1982) Pseudo-speed. *New York State Journal of Medicine*, **1**, 75–75.

Gilbert, R. (1988) Caffeine: the most popular stimulant.In *The Encyclopedia of Psychoactive Drugs* (eds S. H. Snyder & M. H. Lader). New York: Burke Publishing.

Griffiths, R. R. & Woodson, P. P. (1988) Reinforcing properties of caffeine: studies in humans and laboratory animals. *Pharmacology, Biochemistry and Behaviour*, **29**, 419–427.

Harrison, I. (1988) The sale of medicines liable to abuse. *Pharmaceutical Journal*, **May 7**, 600.

Herridge, C. F. & A'Brook, M. F. (1968) Ephedrine psychosis. *British Medical Journal*, *ii*, 160.

Jelley, T. M. (1987) Phensedyl abuse (letter). *British Journal of Psychiatry*, **151**, 418–419.

Lader, M. & Bruce, M. (1986) States of anxiety and their induction by drugs. *British Journal of Clinical Pharmacology*, **22**, 251–261.

Lake, C. R. & Quirk, R. S. (1984) CNS stimulants and the look-alike drugs. *Psychiatric Clinics of North America*, 7, 689–701.

Lambert, M.T. (1987) Paranoid psychosis after abuse of proprietary cold remedies *British Journal of Psychiatry*, **151**, 548–550.

Pugh, C. R. & Howie, S. M. (1986) Dependance on pseudoephedrine (letter). *British Journal of Psychiatry*, **149**, 789.

Rall, T. W. (1985) Central nervous system stimulants. The methylxanthines In *The Pharmacological Basis of Therapeutics* (7th edn) (eds A. G. Gilman, L. S. Goodman, T. W. Rall, *et al*), pp. 589–603. New York: Macmillan.

Siegal, R. K. (1980) Cocaine substitutes (letter). *New England Journal of Medicine*, **302**, 817–818.

Whitehouse, A. M. & Duncan, J. M. (1987) Ephedrine psychosis rediscovered. *British Journal of Psychiatry*, **150**, 258–260.

Williams, M. & Jarvis, M. F. (1988) Adenosine antagonists as potential therapeutic agents. *Pharmacology, Biochemistry and Behaviour*, **29**, 433–441.

S. Loosmore, MB, ChB, MRCPsych, *Consultant Psychiatrist, St David's Hospital, Carmarthen, Dyfed*; *D. Armstrong, MB, ChB, MRCPsych, *Senior Registrar in Psychiatry, Leeds Addiction Unit, 40 Clarendon Road, Leeds*

*Correspondence

British Journal of Psychiatry (1990), **157**, 281–284

A Psychiatric Study of Deviant Eating Behaviour among Mentally Handicapped Adults

G. O'BRIEN and A. M. WHITEHOUSE

A study of deviant eating behaviour among mentally handicapped adults in community placements is reported. Those individuals with a psychiatric disorder showed more deviant eating behaviour. Depressed subjects, in particular, showed an excess of the amount eaten and time spent searching for food, as well as the tendency to eat all sweet food presented to them. Non-food pica was uncommon, even among the autistic subjects.

Eating disorders such as anorexia and bulimia nervosa have been studied extensively in psychiatric patients and in the general population. Recent research has also found that deviant eating behaviour is common in adults with dementia (Hope *et al*, 1989; Morris *et al*, 1989). There have been few studies of eating disorder in mentally handicapped people, and these have been single case studies (e.g. Holt *et al*, 1988) or have concentrated specifically on pica, the persistent eating of non-food substances (McLoughlin, 1988). These studies suggest that pica is more common in mentally handicapped children, in those in institutional placements (McLoughlin, 1987) and in autistic subjects (Kinnell, 1985). There is also evidence that pica and associated deviant eating such as 'food pica' (McLoughlin, 1987) – that is, eating inappropriate (e.g. frozen or uncooked) food – carries a considerable physical morbidity, and possibly, mortality in mentally handicapped people (McLoughlin, 1988). However, little is known of either the prevalence of other deviant eating behaviours in mentally handicapped people, or of their clinical correlates.

The present study focused on reports of the observed eating behaviour of 48 mentally handicapped adults, and sought to identify clinical correlates of deviant eating behaviour.

Method

The 48 mentally handicapped adults who live in three community settings were studied. None had previously been in institutional care. The residents in these three settings were matched for their dependency needs, self-care skills and abilities; they required similar supervision levels and were involved in the same kinds of occupational and social activities. All subjects were able to feed themselves. The sample comprised 48 moderately to severely mentally handicapped adults, as defined by ICD-9 (World Health Organization, 1978). There were 28 men (mean age 38.6 years, s.d. 14.1) and 20 women (mean age 39.6 years, s.d. 16.3).

In each of the three settings, a 'keyworker' system operated, so that there was one member of staff who knew each subject well to act as informants in each case. In addition, records in the form of charted observations of behaviour, which were maintained regularly by the keyworkers, were used alongside informants' accounts of behaviour, in order to maximise the information available.

The observed eating behaviour of the subject over the previous 28 days was determined using a semistructured interview, the eating behaviour section of the Present Behavioural Examination – Mental Handicap (PBE–MH). This is based upon the eating behaviour section of the Present Behavioural Examination (PBE) which is a pre-coded semistructured account of the behaviour of elderly adults with organic brain disease (Hope *et al*, 1989; Morris *et al*, 1989). The schedule consists of 28 key items, grouped under the following headings: factors confounding eating; amount eaten; food choice; eating between meals; searching for food; stealing food; requests for food; abundance of food; inappropriate placing of food; eating style; food left in mouth; eating inappropriate food; eating non-food substances; other eating abnormalities; other oral behaviour; amount of alcohol drunk; requests for alcoholic drinks; amount of non-alcohol drink; requests for non-alcoholic drinks; drinking style. Positive scoring on any one of the key items leads to subsidiary questioning, until satisfactory answers are obtained. In scoring behaviour only those behavioural deviations which the interviewer was quite certain had occurred, on the basis of questioning and records of charted observations, were recorded. This interview took between 30 and 90 min to administer.

All eating behaviour items, like other items assessed in the PBE–MH, were chosen on empirical clinical grounds, on the basis of their clinical importance in mentally handicapped people, and their putative determinants: clearly, for example, in this latter respect it was important to record details of availability of and access to food.

TABLE I
Eating behaviour across diagnostic groups

Eating behaviour	*Diagnostic group (ICD–9 codes)*					
	Autism (299.0) n = *15* *(31%)*	*Schizophrenic psychosis (295.3)* n = *2 (4.1%)*	*Hyperkinetic syndrome (314.1)* n = *4 (8.1%)*	*Depression (296.1)* n = *10* *(20.1%)*	*Neurosis (300.0, 300.2, 300.4, 300.7)* n = *7 (15%)*	*No disorder* n = *10* *(20%)*
Amount						
more	4	1	2	5*	2	0
less	4	0	0	0	1	0
Food choice 'different'						
sweet	1	0	1	4*	2	0
pica	2	0	0	0	0	0
no vegetables	1	0	0	0	0	0
random	1	1	0	0	0	0
total	5	1	1	4	2	0
Eating between meals	6	1	1	4	3	1
Searching for food	6*	1	2	5*	2	0
Abundance of food						
sweet	4	0	0	6*	4	1
other	3	0	3	4	3	2

*$P<0.05$ (all statistics: Fisher's exact test; significance levels by comparison with 'no disorder' group).

Psychiatric assessments were made using a semistructured clinical interview which in turn was used to make clinical psychiatric diagnoses according to ICD–9. This interview was based on that developed by Gillberg (1986) for use in mentally handicapped young adults, and takes approximately 20–30 min to administer. The psychiatric diagnoses were made by one of the authors (GO'B) who is a specialist psychiatrist in mental handicap, as previous reports indicate that, while psychiatric diagnoses are both possible and appropriate in mentally handicapped people with behaviour disorders these diagnoses require specialist orientation (Day, 1985).

Results

Table I shows the numbers of subjects with extreme scores on the principal eating behaviours considered. Here, eating 'more' refers to people who habitually ate at least one-and-a-half times as much as any normal person might be expected to in any circumstance; a 'different' choice of foods refers to people whose choice of food is clearly and habitually either towards or away from the foods detailed in Table I; 'searching for food' refers to people who are constantly looking for food, and who try locked doors etc. to find it; the 'abundance' item refers to people who ate all of the type of food in question, e.g. sweet food, which was placed in front of them or was available to them.

Other data concerning eating behaviour such as physical factors confounding eating, inappropriate placing of foods, food left in mouth, amount of alcohol or other drink taken and inappropriate use of eating utensils did not reveal any interesting or significant findings. On one item, 'eating inappropriate food', which corresponds to food pica (McLoughlin, 1987), seven individuals scored positively, but these were widely distributed over all of the diagnostic groups.

The autistic subjects showed an excess of 'searching for food'. Interestingly, only two cases of non-food pica were found. All other significant findings were in the depressed group of subjects, who showed an excess of eating substantially more food, choosing sweet foods, searching for food, and scoring on the 'abundance' factor for sweet foods, when compared with those who had no psychiatric disorder. The data were also analysed with respect to a number of other important clinical variables including age, sex, use of anticonvulsant medication, antidepressant medication and psychotropic medication, physical illness and motor disability. None of these variables or combination of variables (e.g. by considering subjects who were taking major transquillisers and/or antidepressant medication ($n = 11$) which both might promote appetite for carbohydrate or sweet food) accounted for the findings in respect of the depressed subjects.

Discussion

These subjects were not selected to be representative of all mentally handicapped people. One of the houses specialised in dealing with autistic people (these need close and careful supervision) and the remaining subjects from the other two settings were matched with the autistic subjects in terms of their dependency. The house specialising in autism was included because previous reports had suggested that deviant eating, in particular pica, might be common in autistic subjects (Kinnell, 1985). We did not find this in our study. Interestingly, Kinnell's data were retrospective, whereas our data refer to the immediate 28-day period, and is thus a good indicator or the individual's present state. However, our autistic subjects did score highly on the 'searching for food' item. It is possible that this food-searching behaviour in autistic adults may be an adult counterpart of pica as reported in autistic children (McLoughlin, 1987).

The higher level (80%) of psychiatric disorder we found compared to other studies is probably an artefact of our small and biased sample. For, while researchers have previously reported prevalences of psychiatric disorder in mentally-handicapped people of 15–64%, most agreed that the range is probably 40–50% (e.g. Day, 1985; Lund, 1985). In other respects, and particularly phenomenologically, the psychiatric disorders in our sample were consistent with those of other studies (e.g. Day, 1985; Lund, 1986). The two schizophrenic subjects experienced both auditory and visual hallucinations, neither was epileptic, and they held simple and vivid paranoid delusions. The depressed group was characterised by lethargy, apathy, inability to enjoy activities and, in five of the verbal cases, recent suicidal talk. It was striking to find four adults with a diagnosis of hyperkinetic syndrome. This is a group which requires further study. The low prevalence of neurosis is consistent with other studies (e.g. Corbett, 1979).

It was striking to find a pattern of overeating in the depressed group, and that this was mostly towards carbohydrate and sweet foods. There is an immense literature on the relationship between affective disorder and eating disorder, reviewed by Swift *et al* (1986). Overeating is known to occur in some depressed individuals but poor appetite and weight loss are, of course, much more common. In this respect, Paykel (1977) found that, in a varied sample of depressed patients, 66% had decreased appetite while only 14% had increased appetite. This compares to 50% of our small, highly selected, mentally-handicapped subjects who showed this pattern of overeating.

There are at least three possible explanations of our findings. First of all, severe behaviour problems of many types are known to occur in mentally-handicapped people when they are not adequately stimulated and involved in appropriate activities.

We feel that this is unlikely to explain our findings since this alone cannot explain how the findings have arisen with respect to depression. A second more plausible theory is that we have found a group of mild, but significantly depressed individuals. This is in line with Paykel (1977) who showed that overeating is more common in mildly depressed subjects. However, whereas Paykel reported that depressed women of normal intelligence were more likely to overeat than men, our study did not show any such sex difference. A third possibility, not mutually exclusive from the second, is that some biological dysfunction may underlie the lack of sateity which our depressed subjects showed towards carbohydrate food. Interestingly, this has now also been discussed with respect to a similar pattern of insatiable overeating in elderly demented adults (Hope *et al*, 1989; Morris *et al*, 1989).

In conclusion, while pica was uncommon in the mentally handicapped adults in our study, a pattern of carbohydrate overeating was more common among those subjects with depression. These findings, if repeated in a larger epidemiological study (now in progress in Cambridge) suggest that overeating may be more common in the depressed mentally handicapped adult than in the adult of normal intelligence who is depressed.

Acknowledgements

We are grateful to Drs A. Hope and C. Fairburn for their kind co-operation throughout the development and use of the Present Behavioural Examination – Mental Handicap

References

CORBETT, J. (1979) Psychiatric morbidity and mental retardation. In *Psychiatric Illness and Mental Retardation* (eds F. E. James & R. P. Snaith). London: Gaskell Press.

DAY, K. (1985) Psychiatric disorder in the middle-aged and elderly mentally handicapped. *British Journal of Psychiatry*, **147**, 660–667.

GILLBERG, C., PERSSON, E., GRUFMAN, M., *et al* (1986) Psychiatric disorder in mildly and severely retarded urban children and adolescents: epidemiological aspects. *British Journal of Psychiatry*, **149**, 68–74.

HOLT, G. M., BOURAS, N. & WATSON, J. P. (1988) Down's syndrome and eating disorders: a case study. *British Journal of Psychiatry*, **152**, 847–848.

HOPE, R. A., FAIRBURN, C. G. & GOODWIN, G. M. (1989) Increased eating in dementia. *International Journal of Eating Disorders*, **8**, 111–115.

KINNELL, H. G. (1985) Pica as a feature of autism. *British Journal of Psychiatry*, **147**, 80–82.

LUND, J. (1985) The prevalence of psychiatric morbidity in the mentally handicapped adult. *Acta Psychiatrica Scandinavica*, **72**, 563–570.

—— (1986) Behavioural symptoms and autistic psychosis in the mentally retarded adult. *Acta Psychiatrica Scandinavica*, **73**, 420–428.

MCLOUGHLIN, I. J. (1987) The picas: a study of their aetiology, classification and the medical problems they cause. *British Journal of Hospital Medicine*, **37**, 286.

—— (1988) Pica as a cause of death in three mentally handicapped men. *British Journal of Psychiatry*, **152**, 842–845.

MORRIS, C. H., HOPE, R. A. & FAIRBURN, C. G. (1989) Eating habits in dementia: a descriptive study. *British Journal of Psychiatry*, **154**, 801–806.

PAYKEL, E. S. (1977) Depression and appetite. *Journal of Psychosomatic Research*, **21**, 401–407.

SWIFT, W. J., ANDREWS, D. & BARKLAGE, N. E. (1986) The relationship between affective disorder and eating disorders: a review of the literature. *American Journal of Psychiatry*, **143**, 290–299.

WORLD HEALTH ORGANIZATION (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

*G. O'Brien, MB, ChB, MRCPsych, *University Lecturer and Consultant in the Psychiatry of Mental Handicap, Ida Darwin Hospital, Fulbourn, Cambridge CB1 5EE*; A. M. Whitehouse, BM, MPhil, MRCPsych, *Consultant Psychiatrist, Carlton Hayes Hospital, Leicester, formerly Clinical Lecturer in Psychiatry, University Department of Psychiatry, Addenbrooke's Hospital, Cambridge*

*Correspondence

British Journal of Psychiatry (1990), **157**, 284–287

Firesetting in an Adolescent Boy with Asperger's Syndrome

IAN PAUL EVERALL and ANN LECOUTEUR

Firesetting in children and adolescents is commonly associated with other antisocial acts that comprise conduct disorders. Asperger's syndrome is a rare pervasive developmental disorder. In the case presented we argue that the firesetting arose from the disabilities inherent in Asperger's syndrome. This also indicates that antisocial acts may be symptoms in other psychiatric syndromes as well as a specific conduct disorder.

Asperger first described an "autistic personality disorder" of childhood in 1944. This disorder has been variously named over the intervening years, pointing to the uncertainty of its nosological status as a distinct disorder (Tantam, 1988*a*). It has been described as Asperger's syndrome, a variant of normal personality, a schizoid personality (Wolff & Cull, 1986), or even a mild form of infantile autism (Wing, 1981). In ICD–10 (World Health Organization, 1987), it is however included as a separate category within the group of pervasive developmental disorders.

The clinical features (Wing, 1981) are a qualitative impairment of the ability to interact socially, a profound lack of empathy and an inability to recognise and respond to social nuances such as the gestures and expressions of others. These difficulties are similar to autism but Asperger's syndrome differs by having little delay in language. Use of expressive language can be odd with speech being pedantic, repetitive and circumstantial; the delivery monotonous and inexpressive. There is also a restricted and repetitive repertoire of interests and activities including play which lacks fantasy elements (Mawson *et al*, 1985).

Similarly, there are attempts to formulate the characteristics of children who set fires. Jacobson (1985*a*,*b*) demonstrated that firesetting is far more common among boys in lower social classes and that there is a bimodal distribution with peaks at ages 8 and 13 years. The younger group tend to set fires at home on their own whereas the adolescent group usually set fires in a peer group and away from home. The data consistently show these boys to have more disturbed relationships and to be more aggressive. Jacobson reported 73.5% of the firesetters as conduct disordered, the diagnoses of the remaining subjects including hyperkinetic syndrome, adjustment reaction or neurotic and emotional disorders. The findings are supported by Showers & Pickrell (1987) who described 68% of their firesetter group as conduct disordered. Forensic complications have been noted in Asperger's syndrome: Wolff & Cull (1986) mentioned two boys who had raised fires and Wing (1981) cites four patients performing antisocial acts, "one of whom injured another boy in the course of his experiments on the properties of chemicals". Mawson *et al* (1985) discussed a 44-year-old man with a history of violence and a diagnosis of Asperger's syndrome. Apart from these, the literature citings of Asperger's syndrome and associated antisocial acts are rare. The case presented is an adolescent boy with Asperger's syndrome and a one-year history of firesetting.

Case report

DH is a 17-year-old who first received the diagnosis of Asperger's syndrome at the age of ten years. His one-year history of firesetting began during his last year at school, a time when there was uncertainty regarding his next placement. As a consequence of his firesetting he was expelled from school at age 16 years. Initially, the fires were "small experimental bracken fires". However, the fire brigade attended on three occasions. DH progressed to setting hay and straw stack fires, admitting to at least nine of these. He described how he carefuly planned each fire, choosing the stack during the day and then, late at night, leaving his parents' home taking care not to disturb the household. He always walked to the stack, used matches to start the fire and would watch the blaze from a safe distance to avoid detection. He was finally apprehended when, for the first time, he impulsively lit a fire during the day.

DH is an only child. Both parents are well and there is no family history of developmental disorders or mental illness. His father is a 49-year-old helicopter pilot who works for long periods away from the family home. His mother, a 52-year-old housewife, has taken the major responsibility for her son's care. She admitted that she has found her son's firesetting and subsequent caring for him full-time at home very distressing. When pregnant with DH, his mother suffered pre-eclampsia and was admitted to hospital for three weeks prior to induction at 37 weeks. DH was unresponsive at birth, required oxygen and was separated from his mother for approximately two days.

Developmental history

DH's early development was apparently normal. By age two years he was using two- or three-word phrases although spontaneously preferring to use isolated words only. His vocabulary is described as good and his use of tenses, grammar and syntax normal. However, his speech has always been pedantic, monotonous in tone and with virtually no spontaneous gestures. Currently, although he has no evidence of some of the speech abnormalities seen in children with pervasive developmental disorders, his use of phrases is idiosyncratic, his comprehension concrete and he has particular difficulties with certain abstractions such as understanding the fact that his aunt is also his father's sister.

Socially, DH has always been 'remote' and isolated, having very few friends. As a young child he refused to talk in the presence of strangers. Throughout his childhood he has had a very limited sense of humour and appeared socially naive for his age. In the last year, however, his behaviour had been described as more appropriate with strangers and his mother reported that he had gained some insight into his social difficulties resulting in him feeling embarrassed at times.

DH's play, again from an early age, has always been unusual. He has used toys in a predictable, stereotyped and repetitive manner, had a number of unusual preoccupations and interests, and virtually never demonstrated pretend or fantasy play. His parents gave numerous examples of the special interests he had had since the age of four years which included a preoccupation with water and plastic pipes to the exclusion of other activities, repeated detailed drawings of square-rigged sailing ships and a fascination with trees and tree felling. In addition, he has always found changes in his daily routine and his preoccupations very distressing.

By the age of five years both parents and teachers were aware of his developmental difficulties. When stressed, particularly at school, his behaviour was described as noisy and disruptive, bizarre and abnormal. He was suspended from one and failed in a number of other primary schools. At the age of nine years DH was first placed in a special educational unit where he received more individual attention in a structured setting. His behaviour improved but he remained socially isolated and odd. He was first referred to the child psychiatric services at the age of eight years and recommended for a Steiner school placement for his special educational needs at the age of eleven years. At this time DH was noted to be fastidious and somewhat obsessive in his behaviour and continued to have particular difficulties with new situations. Fortunately, within this specialised boarding educational provision he made some academic progress, gained in self-confidence, developed some relationships with other children and it was said that some of his unusual behaviour had decreased.

Current assessment

DH presented as a healthy but socially immature young man. He was co-operative but quickly became irritated and annoyed if he was interrupted or felt rushed. He failed to develop a rapport, was clumsy in his social overtures and his manner was at times inappropriate. He was very insistent that he was allowed to tell his story in his own way and at his own pace with an inflexible emphasis on detail and chronological order. His voice was monotonous and affectively flat. His understanding was concrete and simplistic; for example, his response to the proverb "people who live in glass houses should not throw stones" was "if you throw stones at a glass house it will let cold air in and it will spoil the ceilings".

DH appeared to understand the meaning of right and wrong, but his explanation for the firesetting was self-centred with limited understanding of its consequences and little empathy for the distress he might have caused. He was clear, however, that he had planned all his fires, except for the last, and had decided to stop before he was caught. He admitted that he enjoyed watching the flames but could not explain why he had impulsively set his final fire. DH was adamant that he would not set further fires but his explanations were unconvincing.

Detailed psychological assessment demonstrated that his intelligence was within the lower end of the normal range but that he did show difficulties and limitations in his ability to understand the perspective of others His reading skills were age-appropriate but his level of comprehension was significantly behind. These results were in keeping with previous test results.

Discussion

This young man has problems affecting many areas of his life which have been present from an early age. His difficulties include severe impairment of his reciprocal social interactions together with a restricted, stereotyped and repetitive repertoire of interests and activities, and as such can probably best be understood as one of the pervasive developmental disorders of childhood. He has exhibited lifelong eccentricity and social isolation and it is possible that this could be considered as an extreme variant of a normal personality or a schizoid personality disorder. However, until there is further clarification of these different disorders, this young man's difficulties are probably best understood within the diagnosis of Asperger's syndrome (Tantam, 1988*c*).

How should his early disturbance of behaviour and his more recent firesetting history at age 18 years be understood in the context of such a diagnosis? Should these aspects of his psychiatric difficulties be considered separately as a secondary diagnosis of conduct disorder? Both behaviour disturbance and firesetting are seen in children with conduct disorders and, furthermore, Jacobson (1985*a*) indicated that the firesetters were a more severe group of conduct-disordered children. Disturbed behaviour and antisocial acts have also been described in a group of adults with Asperger's syndrome (Tantam, 1988*b*). Tantam noted that such young adults are prone to suffer from a range of additional psychiatric disorders. Among the subjects who exhibited antisocial behaviour, he emphasised their lack of empathy and the apparent unaccountability of many of the antisocial episodes. None of the offences seemed to be the consequence of a special interest. Similarly, Wolff & Cull (1986) in their follow-up study of children with schizoid personality disorder again reported the presence of conduct disorders. Although the incidence of conduct disorders was no different to that of a control group, they showed that, in particular, the subjects' inability to show empathy differentiated them from the controls. Turning to DH, as in the Tantam study, his firesetting did not appear to be related to a special area of interest although it did appear to take up a large amount of his time and he was excited about seeing flames. Furthermore, DH had difficulty understanding the distress he had caused and seemed unconcerned about the consequences of his activities.

DH obviously does have a disorder of conduct. However, there is no history of the antisocial symptoms usually associated with a diagnosis of conduct disorder (Rutter *et al*, 1970). DH's antisocial behaviour does not meet diagnostic criteria which are currently accepted for a secondary diagnosis of conduct disorder. This highlights the current diagnostic difficulties when considering isolated and unusual disturbances of conduct.

Throughout his life DH has consistently exhibited strange behaviour in new and stressful situations which, for example, at the age of nine years, resulted in suspension from school. Perhaps the firesetting can be considered as a prolonged episode of disturbed behaviour consequent upon his uncertainty about his future placement as he approached the end of full-time education. It is important to try to understand the prognostic significance for a young man with a pervasive developmental disorder who clearly shows a history of recurrent episodes of behaviour disturbance; Rutter & Giller (1983), in discussing juvenile delinquency and conduct disorders, commented on the continuity of conduct over time and the diversity of the presenting symptoms. A number of studies have emphasised the importance of aspects of this continuity. Minde & Minde (1977) found that infants showing persistent temper tantrums were more likely to be hostile at school age. Similarly, the most troublesome children at school (West & Farrington, 1973) were more likely to become recidivist delinquents at a later age. Finally, in the analysis of three longitudinal studies, Robins (1978) stated that childhood antisocial acts were a strong predictor for adult antisocial behaviour.

It is clear that conduct symptoms not only cluster as specific disorders but seem also to constitute symptoms in a wider range of other psychiatric disorders. This has been indicated by Hollander & Turner (1985) who estimated that of those adolescents detained by the courts, one-third had borderline, paranoid or schizotypal personality disorders. All of the findings demonstrate the prognostic significance of disturbed behaviour as a predictor of future behaviour (e.g. Rutter & Giller, 1983; Robins, 1978). DH's history, we would suggest, also emphasises the important predictive value of past behaviour. As in the subjects of the Tantum study there is very real concern about DH's inability to consider the effect of his actions on other people. This factor therefore forms an important part of the assessment of his dangerousness. However, both from his history and during his assessment there was evidence that he is able to learn to modify his behaviour, for example as demonstrated in the progress he made in his specialised boarding school. Our present understanding of Asperger's syndrome and its relationship with conduct disorder is limited. Yet for a subject like DH there is evidence that such a socially isolated individual is likely to remain profoundly socially handicapped, to be at increased risk of developing other psychiatric disturbances and to require, certainly into early adult life and probably in the long term, specialised sheltered facilities.

Acknowledgement

The authors wish to thank Professor Rutter for his advice and support during the preparation of the manuscript.

References

Asperger, H. (1944) Die autischen psychopathen im kindersalter. *Archiv fur Psychiatrie und Nervenkranheiten*, 117, 76–146.

Hollander, H. E. & Turner, F. D. (1985) Characteristics of incarcerated delinquents: relationship between developmental disorders, environmental and family factors, and patterns of offence and recidivism. *Journal of the American Academy of Child Psychiatry*, 24, 221–226.

Jacobson, R. R. (1985*a*) Child firesetters: a clinical investigation. *Journal of Child Psychology and Psychiatry*, 26, 759–768.

—— (1985*b*) The subclassification of firesetters. *Journal of Child Psychology and Psychiatry*, 26, 769–775.

Mawson, D., Grounds, A. & Tantum, D. (1985) Violence and Asperger's syndrome. *British Journal of Psychiatry*, 147, 566–569.

Minde, K. & Minde, R. (1977) Behavioural screening in pre-school children – a new approach to mental health? In *Epidemiological Approaches in Child Psychiatry* (ed. P. J. Graham). London: Academic Press.

Robins, L. (1978) Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. *Psychological Medicine*, 8, 611–622.

Rutter, M. & Giller, H. (1983) *Juvenile Delinquency. Trends and Perspectives*. New York: The Guilford Press.

Showers, L. & Pickrell, E. (1987) Child firesetters: a study of three populations. *Hospital and Community Psychiatry*, 38, 495–501.

Tantam, D. (1988*a*) Asperger's syndrome (annotation). *Journal of Child Psychology and Psychiatry*, 29, 245–255.

—— (1988*b*) Lifelong eccentricity and social isolation I. Psychiatric, social and forensic aspects. *British Journal of Psychiatry*, 153, 777–782.

—— (1988*c*) Lifelong eccentricity and social isolation II. Asperger's syndrome or schizoid personality disorder? *British Journal of Psychiatry*, 153, 783–791.

West, D. J. & Farrington, D. P. (1973) *Who Becomes Delinquent?* London: Heinemann Educational.

Wing, L. (1981) Asperger's syndrome: a clinical account. *Psychological Medicine*, 11, 115–129.

Wolff, S. & Cull, A. (1986) 'Schizoid' personality and antisocial conduct: a retrospective case note study. *Psychological Medicine*, 16, 677–687.

World Health Organization (1987) ICD–10. *Draft Version of Chapter V*. Geneva: WHO.

*Ian Paul Everall, BSc, MB, ChB, MRCPsych, *MRC Training Fellow and Honorary Senior Registrar in Psychiatry, Department of Neuropathology, Institute of Psychiatry, De Crespigny Park, London SE5*; Ann LeCouteur, BSc, MBBS, MRCPsych, *Lecturer and Honorary Consultant in Child Psychiatry, Department of Child Psychiatry, Institute of Psychiatry, De Crespigny Park, London SE5*

*Correspondence

British Journal of Psychiatry (1990), 157, 288–290

The Validation of the Edinburgh Post-natal Depression Scale on a Community Sample

LYNNE MURRAY and ANDREW D. CAROTHERS

The Edinburgh Post-natal Depression Scale (EPDS) was validated on a community sample of 702 women at six weeks post-partum using Research Diagnostic Criteria for depression. The estimates of sensitivity, specificity and positive predictive value, being based on a large random sample, offer improved guidelines for the use of the EPDS by the primary care team.

Recent epidemiological studies have consistently reported that 10–15% of women suffer from a new onset of depressive illness in the early months following childbirth (e.g. Cooper *et al*, 1988). However, only a small proportion of these cases are identified as depressed by primary health care teams (Cox *et al*, 1987). This failure of detection arises despite considerable input of medical resources for the benefit of mothers and their infants following delivery.

In order to address the problem of the poor detection rate of post-partum psychiatric disorder, Cox *et al* (1987) developed a 10-item self-report questionnaire to identify depression in post-natal samples, the Edinburgh Post-natal Depression Scale (EPDS). This measure was intended for use by health visitors or other primary care workers in the course of their routine contacts. The questionnaire was validated on a post-natal sample and measures of sensitivity, specificity and positive prediction established. However, the sample used for validation was small ($n = 84$). It was also not randomly constituted in that the large majority had already been identified by their health visitors as potentially depressed. A further study has recently been reported in which the EPDS was administered together with a psychiatric interview, and sensitivity and specificity rates calculated using DSM–III criteria (American Psychiatric Association, 1980) for major depression (Harris *et al*, 1989). However, the findings of this study are difficult to interpret because of certain methodological limitations. As in the original validation exercise of Cox *et al* (1987), the sample was not randomly selected: almost half were included because of hyperthyroidism, a condition which invariably presents with psychological symptoms common to anxiety and depression (Gelder *et al*, 1989). Furthermore, the EPDS was completed only *after* a full psychiatric interview which is very likely to have sensitised the women to depressive symptoms that might not otherwise have been acknowledged.

The usefulness of the EPDS as a screen for post-partum psychiatric disorder therefore remains uncertain. The two published validation studies do not give a clear indication of its utility because their findings are likely to have been influenced by both the particular configuration of symptoms and the base rate of disorders in the populations studied (Williams *et al*, 1980, 1983). To ascertain the accuracy with which the EPDS can identify psychiatric disorder in post-partum community populations, specificity, sensitivity and predictive values need to be established on a large representative sample. A study with this aim was therefore conducted.

Method

Women presenting on the post-natal wards of the Rosie Maternity Hospital, Cambridge, during the period February 1986 to February 1988 were approached with a view to possible recruitment into a study of factors influencing infant development. They were included if they fulfilled the following criteria: married or cohabiting, primiparous, aged 20–40 years, having had a 37–42 week pregnancy, and living in the Cambridge City area. Additionally, the baby was required to have had a birthweight of at least 2.5 kg, no gross congenital abnormality, and not to have been admitted to the special-care baby unit. A total of 702 women satisfied these criteria and were approached. Only nine (1.3%) refused to take part in the study.

When the infants were six weeks old the mothers were sent the EPDS together with a stamped addressed envelope for reply. Six hundred and seventy-four (97.3%) returned completed questionnaires, 28 (4%) of whom expressed inability or unwillingness to continue in the study. Of the remaining 646 (92% of the original sample), all those with an EPDS score ≥ 13 and a random sample of those scoring 10–12 ($n = 142$) were interviewed either by a psychiatrist or a psychologist using the Standardised Psychiatric Interview (SPI; Goldberg *et al*, 1970). Additional items concerning weight loss and appetite were incorporated into the SPI in order to give conformity with Research Diagnostic Criteria (Spitzer *et al*, 1975) for at least a minor definite depressive episode. Approximately one in ten of those with an EPDS score < 10 and with no indication of

TABLE I
Range of EPDS thresholds and the corresponding values of specificity, sensitivity and positive predictive value (all values expressed as percentages)

Threshold, T	*Specificity*	*Sensitivity:*[1]			*Positive predictive value:*[1]		
		minor	*major*	*both*	*minor*	*major*	*both*
6.5	54.7	96.2	99.5	98.0	9.8	11.9	21.7
7.5	65.1	93.2	99.0	96.3	11.6	14.4	26.1
8.5	74.6	88.4	97.8	93.5	13.9	18.0	31.9
9.5	82.3	81.6	95.8	89.3	16.5	22.7	39.2
10.5	88.3	73.2	92.6	83.6	19.3	28.5	47.7
11.5	92.5	63.5	88.0	76.7	21.7	35.1	56.8
12.5	95.7	52.0	81.1	67.7	23.6	43.0	66.7
13.5	97.5	41.5	73.1	58.5	24.6	50.5	75.1
14.5	98.7	30.7	62.7	47.9	24.4	58.3	82.7
15.5	99.3	22.6	53.0	39.0	23.5	64.3	87.8
16.5	99.7	14.5	40.6	28.6	21.6	70.8	92.4

1. Estimates for minor and major depression as well as both categories combined.

previous psychiatric history were also assessed with the SPI ($n = 45$). Both interviewers were blind to the EPDS score at the time the interview took place. Following interview, any episodes of depression that had occurred since delivery were defined according to RDC major (definite and probable) and minor definite episodes. These data were then used to validate the EPDS.

Results

Table I gives a range of EPDS thresholds, along with the corresponding values of the sensitivity (i.e. the proportion of depressed women correctly identified), and specificity (i.e. the proportion of non-depressed women correctly identified) as well as the positive predictive value of the scale (i.e. the proportion of women identified as depressed who are truly depressed). Estimates of sensitivity and positive predictive value are shown for major and minor depression separately, as well as for both categories of depression combined. The values were estimated by logistic regression analysis (see Carothers & Murray, 1990), a method that allows for the fact that at different stratifications of EPDS score, different proportions of women were interviewed. The table shows, for example, that a threshold of 10.5 correctly identifies more than 90% of mothers with major depression and nearly 75% of those with minor depression and it produces a sample with about equal proportions of depressed and non-depressed mothers. Similarly, a threshold of 12.5 correctly identifies over 80% of mothers with major depression and about 50% of those with minor depression; and it produces a sample in which about two-thirds of the women are depressed.

Discussion

The validation data obtained in the present study underline the importance of the EPDS as a tool for the identification of depression in post-partum samples. Furthermore, the high response rate, achieved even with postal administration, is impressive evidence of the scale's acceptability to women in the post-natal period. However, it is of note that estimates of those dimensions that are of most relevance to the health care worker (i.e. sensitivity and positive predictive value) are substantially lower than those reported in the two previously published studies. Using a threshold of 12.5, Cox *et al* (1987) reported a sensitivity of 86% and Harris *et al* (1989) one of 95%. This compares with a sensitivity of 67.7% obtained in the present study. However, in view of the method of sampling in the two earlier studies, their findings are likely to represent overestimates of the true utility of the EPDS. Our data, derived from a large representative community population, are more likely to be an accurate estimate of how the EPDS would perform if used routinely in post-natal clinics, and could therefore serve to provide improved guidelines for its use by health care teams. They could also be relevant to the planning of future studies in which the EPDS is to be used as the principal screening instrument.

Acknowledgements

We thank Ruth Sagovsky and Claire Kempton for their assistance in recruitment of subjects, Peter Cooper for his comments on the manuscript, and the Camilla Samuel Fund and the Medical Research Council for financial assistance. Lynne Murray is supported by a Research Fellowship from the Winnicott Trust.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

CAROTHERS, A. D. & MURRAY, L. (1990) Estimating the prevalence of psychiatric morbidity: the use of logistic regression *Psychological Medicine*, **20**, (in press).

Cooper, P. J., Campbell, E. A., Day, A., *et al* (1988) Non-psychotic psychiatric disorder after childbirth: a prospective study of prevalence, incidence, course and nature. *British Journal of Psychiatry*, **152**, 799–806.

Cox, J. L., Holden, J. M. & Sagovsky, R. (1987) Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. *British Journal of Psychiatry*, **150**, 782–786.

Gelder, M., Gath, D. & Mayou, R. (1989) *Oxford Textbook of Psychiatry*. Oxford: Oxford University Press.

Goldberg, D. P., Cooper, B., Eastwood, M. R., *et al* (1970) A standardised psychiatric interview for use in community surveys. *British Journal of Preventive and Social Medicine*, **24**, 18–23.

Harris, B., Huckle, P., Thomas, R., *et al* (1989) The use of rating scales to identify post-natal depression. *British Journal of Psychiatry*, **154**, 813–817.

Spitzer, R. L., Endicott, J. & Robins, E. (1975) *Research Diagnostic Criteria: Instrument No. 58*. New York: New York State Psychiatric Institute.

Williams, P., Tarnopolsky, A. & Hand, D. (1980) Case definition and case identification in psychiatric epidemiology: review and assessment. *Psychological Medicine*, **10**, 101–114.

——, Hand, D. & Tarnopolsky, A. (1983) The problem of screening for uncommon disorders – a comment on the eating attitudes test. *Psychological Medicine*, **12**, 431–434.

*Lynne Murray, PhD, *Senior Research Associate, Child Care and Development Group, Department of Psychiatry, University of Cambridge, Free School Lane, Cambridge CB2 3RF*; Andrew D. Carothers, PhD, *Senior Statistician, The Medical Research Council Human Genetics Unit, Crewe Road, Edinburgh EH4 2XU*

*Correspondence

British Journal of Psychiatry (1990), **157**, 290–292

Folie à Trois among Two Soviet-Jewish Immigrant Families to Israel

SHMUEL MAIZEL, HAIM Y. KNOBLER and RUTH HERBSTEIN

In two cases of folie à trois, affecting two Soviet-Jewish families who emigrated to Israel, both elderly parents in both cases shared the paranoid delusional beliefs of an only child. Severe trauma in the past and social maladjustment in the present may be among the precipitating factors for the development of the shared paranoid disorder.

Shared paranoid disorders are relatively rare. Among them cases of folie à trois are even rarer. In 1942 Gralnic reviewed the entire English literature that dealt with what he referred to as 'psychosis of association'. Of the 103 cases reviewed, only 11 were instances of folie à trois (Gralnic, 1942). Since then only several additional cases have been described (e.g. Wolff, 1957; Dewhurst & Eilenberg, 1961; Fernando & Frieze, 1985).

Increased rates of paranoid states are commonly found among migratory and immigrant groups. The stresses of adaption to a new language, new jobs and a different culture, and feelings of alienation may provide a partial explanation (Hitch & Rack, 1980; London, 1986). Gruenberg (1957), in his review of socially shared psychopathology, stressed the importance of social grouping in determining how fantasies are distinguished from reality.

The following report describes two cases of folie à trois among Russian-Jewish families who immigrated to Israel in the early 1970s. They are of interest not only because of the rarity of the phenomena, but also because of the inadequate mechanisms these families employed to assist them in coping with the hardships of resettlement in Israel.

Case 1

Mr M, a 45-year-old single man, born a Russian Jew and still living with his two elderly parents, was referred to our out-patient clinic for the first time in 1984. He explained angrily that 'psychosocial terrorism' was being used against him. He told the interviewer that a complicated plot had been initiated against him by his mother's sister, an old-time Israeli citizen. The plot included attempts to poison him, as well as the employment of frightening faces to follow him wherever he turned. For these reasons he refused admission to hospital and would not take medication. M's two elderly parents supported his claim that the mother's sister was persecuting their son, and had demanded monetary compensation from her. The mother actively insisted that her sister had connections with the Israeli security services, and was plotting with them to murder her son. She cited her sister's expressions and interpreted them in a delusional way. The father seemed to be a passive person, strongly influenced by his wife, and agreed to everything she said.

Family history

M's 78-year-old mother was born in a Jewish ghetto in the Southern Ukraine. When she was seven, her father was murdered by bandits during the 1919 Civil War. She remembers her father headless, while her mother collected his brain so that she could bury him in his entirety. From a very young age she felt that she was discriminated against because she was Jewish. She was not allowed to finish school, her family was forced to flee their home town and move to Moscow, and there she was denied higher education.

M's 78-year-old father was born in a small town in White Russia into a large family. When he was 17 he left his family and moved to Moscow, so that he could gain his independence. He had also always felt that he had been mistreated at work because of his Jewishness.

M was an only son. He described a normal childhood in a family that continuously faced economic hardship, and always identified with being Jewish. He completed university and worked as an engineer in the Soviet Union. He had never left home, had few friends and did not have relationships with women.

The family emigrated to Israel in 1973, after being invited by the mother's sister to join her. Ten years later, the parents' knowledge of Hebrew was scant, and M felt more comfortable speaking Russian. After arriving the parents never worked, inspite of a steady work record in the USSR. They led an isolated life, made no friends, and survived on social security. M worked for several years as a technician, but left his job because he felt he was being persecuted. For three years prior to the referral he had not managed to hold a steady job, and spent most of his time in bed complaining that people wanted him dead.

A month after the referral he was admitted to hospital after attacking the treating psychiatrist. He was diagnosed as suffering from a paranoid disorder according to DSM-III criteria (American Psychiatric Association, 1980), was treated with haloperidol, and was released four months later. His separation from the parents made little impact on the paranoid belief system held by all members of the family, including M himself. Both parents exhibited paranoid ideation, but neither suited criteria for a diagnosis of a mental illness.

Case 2

Miss F, a 39-year-old single woman, born a Russian Jew and still living with her parents, was first admitted to our hospital in 1979. During the initial interview she claimed that a neighbour was poisoning the family's flat with gas. She admitted to hearing voices speaking about her, exhibited marked loosening of associations, spoke in neologisms, and her affect was flat. A diagnosis of schizophrenia, paranoid type, was made according to DSM-III criteria. F was brought to the hospital by her parents because of bizarre behaviour at home. Both parents supported the patient's notion that a certain neighbour had been poisoning their flat for the last three years. They reported that four months prior to F's admission to hospital the situation had deteriorated to the extent that the family had moved to a new flat in a different neighbourhood – but to no avail.

F was treated with haloperidol (20 mg/day), but showed only slight improvement. Three months later she was released from the hospital, but did not continue ambulatory treatment. During her time in hospital, both parents continued with their paranoid belief that the neighbour was poisoning their flat. In 1981 she was readmitted with similar complaints. Again her parents supported her claim that the same neighbour was gassing their flat. She was treated and released.

In 1985 she was re-admitted to hospital for the third time. However, in the intervening years her mother had died, and neither she nor her father believed that the neighbour was involved in any foul play.

Family history

F's family came from a small town in White Russia. F's elderly father had lost his first wife and two children when their home was bombed during World War II. In 1947 he married F's mother, and two years later F, their only child, was born. According to her parents, F's development was normal. She completed high-school and then gave singing lessons to children in the neighbourhood. She had never left home, did not socialise and had no relationships with men.

The parents told the interviewer several times that they suffered endless harassment as Jews while in the Soviet Union.

In 1974 the family emigrated to Israel. Even though both parents had worked steadily in the USSR, neither attempted to find a job once in Israel. They had no relatives in Israel and did not make social contacts. They survived on social security. F made one attempt to study at the Jerusalem Academy of Music, but was rejected. After that, she spent her time at home with her parents.

During F's first and second admissions to our hospital both her parents exhibited paranoid ideation, but neither suited the criteria for a diagnosis of a mental illness.

Discussion

Among the cases of shared paranoid disorders described in the literature, those that include all the members of the nuclear family – 'folie à famille' –

are very rare (Cotrell & Vibber, 1930; Waltzer, 1963; Glassman *et al*, 1987). The two cases described here are cases of folie à trois that include all the members of the nuclear family. In both cases two elderly parents and an only child, who has never been able to separate from home, share the paranoid ideation.

Glassman *et al* (1987) review six typical features of families who develop folie à famille: social isolation; dependent and ambivalent family relationship; a repeating crisis situation; an underlying threat or the presence of violent behaviours; a stable family membership over a long time; and the evolution of the delusional beliefs around a dominant family member – the inducer. Most of these features were evident in the two families described. Neither family adjusted well to life in Israel: their knowledge of Hebrew was scant; no member of either worked; they had not made any social contacts with local Israelis or with other Soviet-immigrant families; and they had made limited use of social support systems available to Israeli citizens. In both cases the parents had experienced severe trauma in their formative years. They identified with being Jewish, were discriminated against by the authorities on account of their Jewishness, and in general experienced the Soviet Union as a hostile environment. In both families it could be assumed that the inducer of the folie à trois was the mother, the most influential member. Both families had a long-lasting family membership, with dependent family relationships.

The two families became acquainted with the mental health systems only five years (case 2) or ten years (case 1) after their emigration to Israel. This observation is consistent with Littlewood & Lipsedge's findings (1979) of the duration of stay of immigrants before the onset of symptoms (among West Indian immigrants to the UK).

Based on these observations it could be assumed that the development of folie à trois in these families was precipitated also by the interaction between the hardships of the past and the present with their pre-morbid personality and family structure. Garza-Guerrero (1974) states that in order to cope successfully with immigration and resettlement, it is necessary to undergo a series of internal changes similar to the process of mourning, in which the old culture is abandoned and the threat to one's identity is resolved. Wolff (1957) states that psychosis of association occurs in small families or other social groups which are isolated from the rest of the community, and where isolation from each other is far more threatening than isolation from the rest of society.

It appears that these two families both lacked the internal resources to adjust to a society where they no longer had to survive in a hostile environment of discrimination and suspicion. Inspite of the fact that they moved from a country where they experienced severe discrimination as members of a minority to a country where they were part of the majority, they were unable to forego living in an atmosphere that was not hostile. As a result, the entire family shared the paranoid delusions of their mentally ill child, and, in the terms of Soni & Rockley (1974), shaped the content of the delusions so as to express their social milieu.

Acknowledgements

We would like to thank Dr Arie L. Levitin and Dr Oded Grietzer for their help in the preparation of this manuscript.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM–III). Washington, DC: APA.

Cotrell, S. S. & Vibber, F. L. (1930) Report of simultaneous occurrence of psychosis in all members of a family group. *American Journal of Psychiatry*, 10, 287–291.

Dewhurst, W. G. & Eilenberg, M. D. (1961) Folie à trois: case report. *Journal of Mental Sciences*, 107, 486–490.

Fernando, F. P. & Frieze, M. (1985) A relapsing folie à trois. *British Journal of Psychiatry*, 146, 315–316.

Garza-Guerrero, A. C. (1974) Culture shock, its mourning and the vicissitudes of identity. *American Psychoanalytic Society Journal*, 22, 408–429.

Glassman, J. N. S., Magulac, M. & Darko, D. F. (1987) Folie à famille: shared paranoid disorder in a Vietnam veteran and his family. *American Journal of Psychiatry*, 144, 658–660.

Gralnic, A. (1942) Folie à deux, the psychosis of association. *Psychiatric Quarterly*, 16, 230–263.

Gruenberg, E. M. (1957) Socially shared psychopathology. In *Explorations in Social Psychiatry* (eds A. H. Leighton, J. A. Clausen & R. N: Wilson). New York: Basic Books.

Hitch, P. J. & Rack, P. H. (1980) Mental illness among Polish and Russian immigrants in Bradford. *British Journal of Psychiatry*, 137, 206–211.

Littlewood, R. & Lipsedge, M. (1979) Transcultural psychiatry. In *Recent Advances in Clinical Psychiatry* (3rd edn) (ed. K. Granville-Grossman). Edinburgh: Churchill Livingstone.

London, M. (1986) Mental illness among immigrant minorities in the United Kingdom. *British Journal of Psychiatry*, 149, 265–273.

Soni, S. C. & Rockley, G. J. (1974) Socio-clinical substrates of folie à deux. *British Journal of Psychiatry*, 125, 230–235.

Waltzer, H. (1963) A psychotic family – folie à douze. *Journal of Nervous and Mental Disorders*, 137, 67–75.

Wolff, S. (1957) Folie à trois: a clinical study. *Journal of Mental Sciences*, 103, 355–366.

*Shmuel Maizel, MD, *Department Director, Eitanim Psychiatric Hospital, Hebrew University Medical School, Doar Na Shimshon, Jerusalem 99790, Israel*; Haim Y. Knobler, MD, *Resident in Psychiatry, Eitanim Psychiatric Hospital*; Ruth Herbstein, MSW, *Psychiatric Social Worker, Eitanim Psychiatric Hospital*

*Correspondence

British Journal of Psychiatry (1990), 157, 293

Rabbit Syndrome – A Rare Complication of Long-Term Neuroleptic Medication

D. K. DESHMUKH, V. S. JOSHI and M. R. AGARWAL

A case of rabbit syndrome, a complication of long-term neuroleptic medication, is reported. It is important to differentiate it from tardive dyskinesia and continuous therapy with an antiparkinsonian agent may be required for control of symptoms of rabbit syndrome.

The term 'rabbit syndrome' was first coined by Villeneuve (Sovner & Dimascio, 1977). It is an uncommon, tongue-sparing, orofacial movement disturbance, which is rapid and regular and associated with prolonged use of neuroleptics (Todd *et al*, 1983).

It is important to diagnose tardive dyskinesia differentially from rabbit syndrome by noting the sparing of tongue movements and the response to antiparkinsonian agents in the latter.

Case report

Mr K, a 52-year-old male patient, diagnosed as suffering from schizophrenia since 1969, had been admitted to hospital repeatedly for acute exacerbations of psychotic features. He had been receiving neuroleptics almost regularly for 20 years, on an out-patient basis. On one such out-patient visit, he was found to have orofacial and jaw movements which were rapid, involuntary and rhythmic and were occurring at a rate of 50 cycles/min. There were no involuntary movements of the tongue or of any other part of the body. These movements reduced on voluntary activity. No other signs of extra-pyramidal dysfunction were found.

A diagnosis of rabbit syndrome was made and oral trihexyphenidyl (2 mg t.i.d.) was started and oral trifluoperazine, which he had been receiving, was discontinued. By the third day of starting trihexyphenidyl, his orofacial and jaw movements reduced, disappearing completely by the fifth day. The patient, however, stopped his medication of his own accord and his abnormal orofacial and jaw movements reappeared after four to five days. He was restarted on oral trihexyphenidyl (2 mg t.i.d.) and again his abnormal movements disappeared completely within four to five days. Since then he has been on oral trifluoperazine (5 mg t.i.d.) and trihexyphenidyl (2 mg t.i.d.) and is followed up regularly.

Discussion

Our case illustrates the successful use of oral trihexyphenidyl in controlling the symptoms of rabbit syndrome. The disappearance and reappearance of the symptoms of rabbit syndrome after a period of starting and stopping the antiparkinsonian agent suggests that the syndrome does respond to these drugs, and continuous treatment may be required to control the symptoms. These observations also help to confirm the diagnosis of rabbit syndrome. A favourable response to an antiparkinsonian agent indicates that the pathophysiology of rabbit syndrome may be similar to that of acute forms of drug-induced parkinsonism (Jus *et al*, 1972).

Both rabbit syndrome and tardive dyskinesia are usually seen after a prolonged exposure to neuroleptic agents, in middle-aged or elderly patients. The high frequency of the perioral movements, and the absence of lingual movements are the main features that distinguish rabbit syndrome from tardive dyskinesia since the latter is characterised by less frequent, more irregular movements (Todd *et al*, 1983). The use of intravenous physostigmine has also been described to distinguish between rabbit syndrome and tardive dyskinesia. Physostigmine may worsen the symptoms of rabbit syndrome but reduce those of tardive dyskinesia (Weiss *et al*, 1980). However, we did not use this test.

Recognising rabbit syndrome and differentiating it from tardive dyskinesia is thus important in the proper treatment of orofacial disorder following long-term neuroleptic medication.

References

Jus, K., Villeneuve, A. & Jus, A (1972) Tardive dyskinesia and Rabbit Syndrome during wakefulness and sleep (letter to editor). *American Journal of Psychiatry*, 129, 765.

Sovner, R. & Dimascio, A. (1977) The effect of benztropine mesylate in the rabbit syndrome and tardive dyskinesia. *American Journal of Psychiatry*, 134, 1301–1302.

Todd, R., Lippman, S., Manshadi, M., *et al* (1983) Recognition and treatment of rabbit syndrome: an uncommon complication of neuroleptic therapies. *American Journal of Psychiatry*, 140, 1519–1520.

Weiss, K. J., Cirauto, D. A. & Shader, R. I. (1980) Physostigmine test in rabbit syndrome and tardive dyskinesia. *American Journal of Psychiatry*, 137, 627–628.

*D. K. Deshmukh, *Honorary Associate Professor in Psychiatry, J J Hospital and Grant Medical College, Byculla, Bombay, 400 008, India*; V. S. Joshi, *Resident in Psychiatry, J J Hospital and Grant Medical College*; M. R. Agarwal, *Resident in Psychiatry, J J Hospital and Grant Medical College*

*Correspondence

British Journal of Psychiatry (1990), **157**, 294–303

Correspondence

Editor: Ian Pullen

Contents: The 'new cross-cultural psychiatry'/Impact of participating in research/Genital self-mutilation/Psychiatric sequelae of listeriosis/Aggressiveness, anxiety and drugs/Limitations of double-blind trials/Reviewing reviewers/ROC analysis/The Lomax affair/Brain transplants: myth or monster?/Patterns of attendance at child psychiatry clinics/First-rank symptoms

The 'new cross-cultural psychiatry'

SIR: In his editorial on the 'new cross-cultural psychiatry', Leff's assertion (*Journal*, March 1990, **156**, 305–307) that the disease categories of Western biomedicine are universally valid is mistaken, and his statement about its "manifest success in treating diseases like smallpox" is difficult to sustain in light of the evidence provided by disciplines including medical sociology, anthropology and the history of medicine. Research in these areas points to the particular and ultimately ethnocentric nature of all medical classifications. The positivist notion of medicine as a set of technical interventions divorced from social, political and cultural influences is surely untenable in the late 20th century. That medicine is rather a social institution and functions as such in every society, be it in Africa or Europe, is now widely accepted.

The success of any medical classification has to be seen in this context. The criteria used by Western medicine to judge its own performance are provided by the institutional framework within which it operates. The eradication of smallpox only proves the "manifest success" of Western biomedicine within the confines of a Western ontology and world-view. In addition, if medicine is not simply a set of techniques but rather a social institution, the evaluation of its efforts is a lot more complicated than Leff supposes. For example, in the Kasongo vaccination project in Zaire (Kasongo Project Team, 1981), while a high coverage of measles immunisations was achieved and led to a noticeable reduction in measles mortality, the overall mortality was not affected. The same number of children perished, but from other causes. Was the medical intervention successful? By what criteria do we judge?

Some time ago, Illich (1976) argued that if we look at Western biomedicine as an institution and examine its social and cultural effects in Western countries, we are led to the conclusion that its overall impact is one of iatrogenesis. While one may not agree fully with Illich's conclusions, his work should make us wary of statements about the "manifest success" of Western biomedicine.

The 'new cross-cultural psychiatry' as presented by Littlewood (*Journal*, March 1990, **156**, 308–327) opens the way for the development of a discipline which is genuinely self-reflective and which confronts the ethnocentric nature of our current systems. However, if we continue to pursue an approach based on positivist biomedicine, we are simply ignoring this ethnocentricity, which is ultimately the real problem of our discipline. It will not go away. Aspiring to the "manifest success" of biomedicine should surely not be the goal of psychiatry in the 1990s.

PATRICK J. BRACKEN
JOAN E. GILLER

PO Box 269
Kampala
Uganda

References

ILLICH, I (1976) *Limits to Medicine–Medical Nemesis: The Expropriation of Health*. Harmondsworth: Pelican.

KASONGO PROJECT TEAM (1981) Influence of measles vaccination on survival pattern of 7–35 month old children in Kasongo, Zaire. *Lancet*, *ii*, 764.

SIR: Leff (*Journal*, March 1990, **156**, 305–307) offers a captious view of Littlewood's review of the 'new cross-cultural psychiatry' (*Journal*, March 1990, **156**, 308–327). For instance, Leff cites Littlewood's (1984) paper which, contrary to Leff's interpretation, neither idealises nor romanticises insanity, but rather demonstrates how intellectual innovation can arise from a particular society's reponse to the inversion of normal behaviour by two messianic leaders who experienced episodes of psychosis.

Dr Littlewood's review includes some suggestions on how clinically applied anthropology can, for example, enhance the role of the liaison psychiatrist on psychosomatic wards. This is hardly a Laingian anti-psychiatry enterprise.

Professor Leff fails to do justice to the important conceptual and practical innovations offered by Kleinman (1988). These include the systematic creation of an ethnography of each patient's experience of illness and his or her "explanatory model"; a recognition of the culture-bound nature of the idioms of distress used by laymen to indicate dysphoria; and a heightened awareness of psychiatry's own cultural origins and tacit assumptions and its tendency to systematically minimise cultural differences in the symptoms and interpretation of mental illness.

MAURICE LIPSEDGE

Guy's Hospital
St Thomas Street
London SE1 9RT

References

KLEINMAN, A (1988) *Rethinking Psychiatry From Cultural Category to Personal Experience.* New York: Free Press

LITTLEWOOD, R (1984) The imitation of madness: the influence of psychopathology upon culture. *Social Science and Medicine*, **19**, 705–715.

SIR: Leff (*Journal*, March 1990, **156**, 305–307) gives the wrong impression about what anthropologically-orientated studies in psychiatry are all about.

Professor Leff uses the example of smallpox to criticise an epistemological approach to psychiatry, which he appropriately associates with anthropological works on psychiatry, yet inappropriately implies is a continuation of nihilistic anti-psychiatry. For Professor Leff, vaccination, which Jenner developed from 18th-century English folk beliefs, was successful in eradicating smallpox; Yoruba and Hindu ethnotheories about this disease, however, were not effective and therefore, unlike biomedical categories, did not become dominant. Although his intended meaning is unclear here, especially since he qualifies his argument with examples where success is greater for indigenous therapies of Third World societies, Professor Leff seems to imply a utilitarian justification of psychiatric categories.

This is a strange conclusion to draw from the smallpox case for several reasons. The World Health Organization's (WHO's) eradication campaign was a logistic, administrative and political triumph which drew upon knowledge of local cultures and the WHO's own institutional culture to overcome obstacles, some of which derived from contemporary public health policies. Furthermore, as Marglin (1988) indicates, the eradication of smallpox in the Indian subcontinent had some ironic twists. The traditional Indian practice of variolation, which had a certain measure of efficacy, was outlawed by the British government in India in 1865, and this both worsened the public health situation and contributed to the colonial policy of fostering Indian dependence in the sphere of health. Later on during British rule, the popular resistance to the compulsory vaccinations introduced in some provinces was a surrogate for more widespread political resistance and led to a worsening of the smallpox problem. Moreover, the forced vaccinations in the 1960s and 1970s, which ultimately eradicated smallpox, produced such resistance that its after-effects still reverberate, causing obstacles to public health programs. It was not biomedical categories, then, but cultural values and political interests which fostered both the successes and negative consequences of smallpox eradication campaigns.

The beliefs, rituals and community activities in honour of Sitala – the smallpox goddess – are associated with "their ability to regenerate the community, to create or re-create social consequences" (Marglin, 1988, p. 7) and are part of a Hindu view of health as the *result* of the experience of illness and death, and not as their opposite or enemy. Seen from the perspective of how Indian culture responds to near-death experiences of the aged and the chronically ill, the Hindu conception may be more successful, at least with respect to these experiences, than its biomedical counterpart.

The anthropological approach to psychiatry begins with cultural epistemology because this is useful to understand the indigenous or lay beliefs about illness and how these constrain treatment decisions. Anthropological investigations also show how a health problem is constructed as a technical/professional problem through the application of diagnostic categories and clinical assessment methodologies, and how this creates an 'object' of psychiatric practice. Biomedical categories strip away the illness experience, including the moral condition of suffering, to get at disease pathology. When that reduction is successful, as is the case with many treatable acute infectious diseases, it is an example of a remarkably useful cultural bias. When it is relatively unsuccessful, which is often the case in chronic medical and psychiatric conditions, the outcome is a form of dehumanised care *and* also an impoverished scientific epistemology for medicine/psychiatry.

In my book (Kleinman, 1988), I tried to show how an epistemological approach to psychiatric practice can be a useful method for uncovering and remedying serious biases in our taxonomies, value hierarchies and treatment practices that limit our discipline's utility in developed *and* developing societies. The role of anthropology itself should be to clarify when the 'culture' of psychiatry itself becomes an obstacle to effective and humane treatment and prevention programs. Thereby, anthropologically-oriented psychiatry can contribute toward a kind of emancipatory self-reflection on how to keep our rational technical devices from becoming an iron-cage of reified (and as in much of DSM–III–R, commoditised) classifications whose utility is much less powerful and whose 'object' is all too readily dehumanised.

Cultural epistemology offers a needed complement to epidemiological, clinical and psychometric approaches to cross-cultural comparisons, and, since a knowledge of anthropology is as important in cross-cultural psychiatry as an understanding of, for example, neuroscience, Littlewood (*Journal*, March 1990, **156**, 308–327) should be praised for his anthropological contribution on how cultural meanings affect mental illness and psychiatry.

Arthur Kleinman

Department of Anthropology
Harvard University
William James Hall
Cambridge, Massachusetts 02138

References

Kleinman, A. (1988) *Rethinking Psychiatry From Cultural Category to Personal Experience*. New York· Free Press.

Marglin F A (1988) *Smallpox in Two Systems of Knowledge* Helsinki. UNU/WIDER Working Paper.

Sir: In my editorial (*Journal*, March 1990, **156**, 305–307) I did not intend to slight the valuable and innovative achievements of the 'new cross-cultural psychiatry' which Littlewood (*Journal*, March 1990, **156**, 308–327) detailed in his extensive review. Rather than underlining the points made in the review, I focused on the issue of the validity of psychiatric disease categories, which the new approach has brought into question. On reading Professor Kleinman's letter, I began to wonder whether I had set up Dr Littlewood as a straw man. However, behind this possibly spurious target stand solid ranks of critics, both within and outside the profession, who have seized as ammunition the notion that psychiatric categories are cultural constructions with no validity outside the realm of biomedicine.

The point I was attempting to illustrate with the example of smallpox eradication has been largely misinterpreted by Professor Kleinman and Drs Bracken & Giller. My argument does not rest on the overall impact on the health of a society, vitally important though that is. I maintain that the response of a condition to a remedy that is postulated to ameliorate or cure that condition and no other constitutes a validation of the disease category. If the same condition shows the same responsiveness across cultures, then that is evidence for the cross-cultural validity of the disease category in question. In short, I do support a 'utilitarian justification' of disease categories, including those constructed within psychiatry.

If this criterion for validity were confined to Western biomedicine, then we would be trapped within a tautology from which escape was impossible. However, I agree with Dr Haldipur that this approach is not exclusive to Western biomedicine. There is no doubt that traditional healers have discovered specific remedies for conditions that biomedicine recognises as diseases categories (e.g. quinine for malaria, Rauwolfia for psychoses). It is inconceivable that they could have established this link between condition and remedy without applying the principle of utility, and using it to select the patterns of presentation that were responsive. For healers and clients the acid test of a remedy is its effectiveness as a cure. As Waxler (1977) writes from her experience in Sri Lanka, "No family lingers long with any treatment person; one or two visits, one or two bottles of medicine are enough to convince them that the treatment cures or does not cure. If it is ineffective they move on; if it is effective the patient is 'cured' and needs no more".

It is here, I believe, that the doctor and the anthropologist face an ideological divide. As Kleinman affirms, an anthropologist is concerned with the value to society of rituals which "regenerate the community", even though they may be totally ineffective in helping the sick individual. However, the doctor's prime duty and responsibility is to that individual, even if she or he takes preventive measures at a societal level (e.g. by ensuring a supply of clean water). The doctor should be sensitive to cultural values when instituting preventive or curative measures, such as vaccination, but in contrast to the anthropologist, his or her paramount aim is to alleviate suffering. The construction of hypothetical disease entities based on a 'notion of pathology' is an essential stage in achieving this aim, a principle recognised by Western doctors and traditional healers alike.

J. Leff

MRC Social and Community Psychiatry Unit
Institute of Psychiatry
De Crespigny Park
London SE5 8AF

Reference

Waxler, N. E. (1977) Is mental illness cured in traditional societies? A theoretical analysis *Culture, Medicine and Psychiatry*, 1, 233–253

SIR: There are conceptual and methodological problems with Littlewood's 'new cross-cultural psychiatry' (*Journal*, March 1990, **156**, 308–327), just as there are with the more traditional cross-cultural psychiatry (Haldipur, 1979). I shall adumbrate an epistomological approach to the new cross-cultural psychiatry similar to that taken by Dr Littlewood to psychiatry.

(a) Culture is difficult to define without tautology, as Dr Littlewood himself acknowledges (Appendix, point 3). Not only are there trans-Atlantic differences in the approach, but there may be over a hundred definitions of culture. It is often a blanket term used to designate a variety of environmental factors.

(b) Although cultural and societal norms and values exist, conformity or non-conformity to these by individuals usually calls for psychological explanations (Homans, 1967).

(c) There is a 'category error' in stating that culture *influences* or *causes* us to consider certain types of behaviour as normal or abnormal: among other ideas, those about what is normal behaviour *is*, by most definitions, culture. It is difficult then, to separate ideas about behaviour from culture of which it is a part.

(d) We are told that if the new cross-cultural psychiatrists had their way, research in various communities would be done by anthropologists and not psychiatrists in order to obviate any influence of Western psychiatric education in perceptions of abnormality. However, anthropologists cited by Dr Littlewood appear to focus their interest on larger groups such as ethnic communities, tribes or even nations. Surely, individuals live in families. And might it not be safe to assume that the norms and values inculcated by families may be equally important? On occasions, these norms and values may run counter to those of the larger group, such as in the case of ethnic minorities. An anthropologist truly interested in carrying out Leff's mandate (*Journal*, March 1990, **156**, 305–307), while studying the larger community, may have to acknowledge that individuals belong to various concentric groups or organisations, and that it is difficult to separate and weigh the relative importance of these.

(e) A perusal of references cited by the two authors shows that a number of them are studies in non-Western countries or of ethnic minorities living in the West. It makes one wonder if for new cross-cultural psychiatrists, 'culture' stopped somewhere this side of Suez. What is even more troubling is the predilection for the non-literate sections of those societies. This could have been a relatively harmless preoccupation, except that sweeping generalisations are often based on observations of a small section of the community. It is as if one were to write about healing practices in north-eastern United States by observing Christian Scientists in Boston, without any reference to practices of the majority. The description of Mahanubhav healing centres of western India reveal interesting practices and rituals; but they exist side by side with Ayurvedic (Obeyeskere, 1977) and allopathic medical practices. The former, indigenous to the region, has a sophisticated classificatory system, not too dissimilar from our own (Haldipur, 1989). Thus, to characterise the more scientific modern system of biomedicine as entirely Western, and its adoption by the Third World as 'psychiatric imperialism', is probably unjustified.

Finally, it may be worth recalling the words of the well known anthropologist Claude Levi-Strauss in *Tristes Tropiques*: "I had looked for a society reduced to its simplest expression That of Nambikwara was so far reduced that I found only men there".

C. V. HALDIPUR

Psychiatry Service
800 Irving Avenue
Syracuse, New York 13210

References

HALDIPUR, C V. (1979) The idea of "cultural" psychiatry. a comment of the foundations of cultural psychiatry. *Comprehensive Psychiatry*, **21**, 206–211

—— (1989) Psychiatric nosology and taxonomy in ancient India *Acta Psychiatrica Scandinavica*, **80**, 148–150.

HOMANS, C. G. (1967) *The Nature of Social Science* New York Harcourt, Brace & World.

OBEYESEKERE, G. (1977) The theory and practice of psychological medicine in the Ayurvedic tradition. *Culture Medicine Psychiatry*, **1**, 155–181.

SIR: Dr Haldipur conflates my paper (*Journal*, March 1990, **156**, 308–327) with that of Leff (*Journal*, March 1990, **156**, 305–307) who is, on the substantive points, saying the opposite of myself.

In as much as I can grasp the comments made on my own paper, I agree about the problem of defining culture: indeed, I refrain from attempting such a task, merely citing Geertz' not unuseful statement. To Cisatlantic anthropologists, 'culture' and 'society' are synonymous. The new psychiatry would deny, however, that conformity to social norms is best approached through the procedures of individual psychology, for the norm and its inversion both have their social meaning, as legitimate from the scientific perspective as any other, as simply elements in a complex web of interactions in a society. I certainly agree that if we say that culture influences behaviours (or actions as anthropologists would rather say), then we are indulging in a category error. 'Culture', however we define it, represents a society with all its conflicts and contradictions and its relationship with other societies; the boundaries are arbitrary. I am increasingly wondering if *political* is not quite simply a better term than *cultural.*

I am not altogether sure what families are, nor am I convinced that they are things which people generally live in and which ultimately determine their lives: socialising institutions doubtless, but not socially determining institutions.

ROLAND LITTLEWOOD

Department of Anthropology
University College London
Gower Street
London WC1E 6BT

Impact of participating in research

SIR: Ben-Arie *et al*'s study (*Journal,* January 1990, **156,** 37–39) prompted us to conduct a similar one, examining the effect of research on psychiatric admissions and other psychiatric contacts, using a more specific group (depressed in-patients) and over a more immediate follow-up period.

We have re-analysed the data for 57 depressed in-patients who participated in one of the Nottingham electroconvulsive therapy (ECT) studies (Gregory *et al,* 1985). Using the Nottingham case register, we have compared their outcome at six months with that of 38 other patients who satisfied the entry criteria for the ECT study but did not enter, mainly due to lack of consent, and who were thus not followed up by the research team. In this example, as in that of Drs Ben-Arie *et al,* the clinical and research teams were separate.

We found no statistically significant differences between the two groups as regards the number and lengths of hospital or day-care admissions. The number of out-patient and other contacts were also similar for both groups.

These results suggest that participating in research did not have a significant effect on short-term outcome for these depressed in-patients. Although the two groups were not randomly allocated, demographic variables and mean index admission scores on the MADRS and HAM rating scales for the two groups were not significantly different. In contrast to the study of Drs Ben-Arie *et al,* the mean number of previous admissions and the mean length of the index admission were also similar for both groups.

It may be that the effect of research on admissions only becomes evident after six months. However, another more intriguing possibility is that the effect of research may be inversely proportional to the number of projects undertaken in any given clinical setting. The demographic profile of Nottingham lends itself to clinical research, and the frequency of such work here may have acted to desensitise patients and clinicians to its disturbing effects on presentation and clinical practice.

DENIS O'LEARY
ALAN LEE

University Hospital
Queen's Medical Centre
Clifton Boulevard
Nottingham

DAVID GILL

Mapperley Hospital
Nottingham NG3 6AA

Reference

GREGORY, S., SHAWCROSS, C R & GILL, D. (1985) The Nottingham ECT study a double-blind comparison of bilateral, unilateral, and simulated ECT in depressive illness. *British Journal of Psychiatry,* 146, 520–524.

Genital self-mutilation

SIR: Walter & Streimer (*Journal,* January 1990, **156,** 125–127), in a case report of genital self-mutilation in a non-psychotic adult male, indicated that "The patient's father ... was remembered as punitive and distant. The patient's mother was more available but was perceived as devaluing and affectionless", and stated that "The nature and dramatic culmination of the patient's dysmorphophobic symptoms may be understood in terms of a childhood during which he was demoralised, emasculated, and deprived of recognition". Finally, they emphasised that non-psychotic genital self-mutilation, while uncommon, may not be as rare as is generally stated.

However, the authors did not mention the cases of genital self-mutilation which occurred during the ancient mediterranean rites. In 1922, Frazer reported that the Great Mother Cybele's worship required from followers a ceremonial genital self-mutilation in recollection of the Attis' mythological experience.

Men who intentionally mutilate their own genitals are likely to be psychotic, and their behaviour may be considered as an acute psychotic breakdown in the context of a schizophrenic regression. Indeed, it should be conceived as an attempt to return to the mother's womb (Roccatagliata, 1982).

Roth & Ball (1963) found that, in a high proportion (94%) of male cases of transsexuality, there had been extreme dependence on and a strong preference for the mother of the family, and the father of the majority had either been absent for long periods during the formative years (59%) or insignificant in the family setting, or had had a personality marked by coldness, hostility or psychopathic traits (77%). The child had thus been driven into an exclusive relationship with the mother, even when she had herself possessed some abnormal traits.

Genital self-mutilation is a psychotic disorder, although its relationship with dysmorphophobia (Morselli, 1886) is debatable (Birtchnell, 1988). The patient's familiar clinical picture, as reported by the authors, is congruent with this hypothesis.

PAOLO NOVELLO
ALBERTO PRIMAVERA

Department of Neurology
University of Genoa
Via De Toni 5
16132-Genoa, Italy

References

BIRTCHNELL, S. A. (1988) Dysmorphophobia – a centenary discussion. *British Journal of Psychiatry*, **153**, (suppl 2), 41–43

FRAZER, J. G. (1922) *The Golden Bough A Study in Magic and Religion*. London: Macmillan

MORSELLI, E. (1886) Sulla dismorfofobia e sulla tafefobia. *Bollettino delle Scienze Mediche di Genova*, VI, 100–119.

ROCCATAGLIATA, G. (1982) *Il Culto della Grande Madre e la Psicosi* (ed. R. Vizioli). Roma Il Pensiero Scientifico

ROTH, M & BALL, J. R. B. (1963) Psychiatric aspects of intersexuality. In *Intersexuality* (eds A. J Marshall & C. N Armstrong) London Academic Press

Psychiatric sequelae of listeriosis

SIR: Duncan recently reviewed the scant literature on psychiatric syndromes caused by central nervous system (CNS) infection with listeria monocytogenes (*Journal*, June 1989, **154**, 887), which is in sharp contrast to the abundant reports describing neurological complications (Pollock *et al*, 1984). Because of the growing importance of listeriosis in the differential diagnosis of psychiatric syndromes, especially in immunocompromised hosts, we would like to point out two further psychiatric sequelae not mentioned by Dr Duncan and report the case history of one of our patients.

Orland & Daghestani (1987) described a patient in whom several catatonic episodes occurred in the course of a listeria meningo-encephalitis. This syndrome (not included in the report of schizophrenia-like listeria psychosis by Timofeyeva *et al*, 1953) was accompanied by frontal lobe electroencephalogram (EEG) abnormalities, and subsided after antibiotic treatment. Secondly, features of a chronic psycho-organic syndrome after CNS listeriosis might often escape diagnosis, as both patients and their families will tend to neglect minor changes which can appear insignificant in the aftermath of a highly lethal disease. In adults, Seeliger (1955) mentioned incomplete recovery after listeriosis with persistent disturbance of memory and speech.

Here, we report the case of a 68-year-old male patient who, after a holiday in South America, had fallen ill with a listeria meningo-encephalomyelitis. In the acute phase of the illness he suffered extensive right-sided neurological deficits which recovered after adequate treatment. A few months later the patient complained of a loss of intellectual abilities with impaired short-term memory, difficulty in concentration, and a generalised apathy and lack of interest and drive. He was angry that these symptoms did not improve as did his physical complaints. He proved unable to resume his occupation in senior management. Cerebrospinal fluid showed no evidence for chronic inflammation, cranial computerised tomography and magnetic resonance imaging revealed several small lesions in the cerebral medulla around the ventricles, EEG showed generalised slowing, and single photon emission computerised tomography was unremarkable. The formerly highly intelligent patient achieved only an average IQ on the Hamburg-Weschler Intelligence Test for Adults, and was markedly cognitively slowed in several tests. Minor deficits in short-term memory, problem solving and attention were regarded as severe by the patient when he compared them with his pre-morbid abilities. There was no evidence for a depressive disorder. Although an early dementia secondary to a long history of arterial hypertension cannot definitely be excluded, we believe that the described syndrome is a late complication of the listeria meningo-encephalitis.

M. KELLNER
A. SONNTAG
F. STRIAN

Max-Planck Institute for Psychiatry
Kraepelinstraße 10
D-8000 Munich 40

References

ORLAND, R. M & DAGHESTANI, A. N. (1987) A case of catatonia induced by bacterial meningoencephalitis. *Journal of Clinical Psychiatry*, **48**, 489–490.

POLLOCK, S S., POLLOCK, T. M & HARRISON, J. G. (1984) Infection of the central nervous system by listeria monocytogenes: a review of 54 adult and juvenile cases *Quarterly Journal of Medicine*, **211**, 331–340.

SEELIGER, H. (1955) *Listeriose. Beitrage zur Hygiene und Epidemiologie*. Leipzig: Heft 8.

TIMOFEYEVA, A., SHKURKO, E. D. & UDALTSOVA, M. S. (1953) On listeria psychosis. *Zhurnal Neuropatologii i Psikhiatrii imeni S.S. Korsakova*, **53**, 625–631.

Aggressiveness, anxiety and drugs

SIR: Kirov (*Journal*, December 1989, **155**, 846) equates the control of anxiety and aggression: drugs which control anxiety control aggression, and those which aggravate anxiety aggravate aggression.

Clinically, this is often not true, particularly in the field of child protection from abusive carers. Alcohol or tranquillisers can make the parent/carer more dangerous. The mechanism for this is that the parent already hates, resents, or is frustrated by the dependent child. Theoretically, the aggression itself could be dulled by the anti-anxiety drug (alcohol, benzodiazepine or barbiturate). However, cortical restraint is also damped down, releasing or aggravating whatever aggressive propensities remain.

This throws a caution on Professor Kirov's last paragraph, in which optimism is expressed that anti-anxiety drugs will control aggression.

JACK E. OLIVER

Burderop Hospital
Wroughton
Swindon, Wilts SN4 0QA

Limitations of double-blind trials

SIR: Newcombe (*Journal*, February 1990, **156**, 282), in his letter on double-blind trials, continues to defend randomised controlled trials in psychiatric research (Newcombe, 1988), without apparently recognising their limitations (Kramer & Shapiro, 1984). In particular, he does not accept the fallibility of the double-blind and criticises Oxtoby *et al*'s (1989) suggestion that the ability of participants to guess their drug status should be used as a retrospective criterion to exclude certain results. Dr Newcombe's caution about this latter suggestion is justified, although the recording of guesses of whether patients were taking active drugs or placebo can be of value. As a recent example, Marks *et al* (1988) found that assessors' guesses after the end of treatment were mostly right. They did not assess the blindness of patients but suggested that such checks might yield similarly sobering data. Moreover, Oxtoby *et al* (1989) advocate reworking results when the double-blindness has been disproven, and the question then arises of why the trial was not double-blind.

The breaking of the double-blinding on occasions has been interpreted as the explanation for a positive trial result. For example, Karlowski *et al* (1975) found that ascorbic acid seemed to reduce the duration of a common cold, but these differences were eliminated when taking into account the correct guesses of medication. Practically, it is difficult to know whether the issues that Dr Newcombe raises about upsetting the randomisation and similar problems would be relevant to this study.

Perhaps a more appropriate conclusion to draw from this debate is that clinical trials are unlikely to be definitive in the scientific sense that Dr Newcombe would like. Interpretation of results is inevitably important, which may explain why there is so much controversy about the effectiveness of psychiatric treatment.

D. B. DOUBLE

Department of Psychiatry
University of Sheffield
Northern General Hospital
Sheffield S5 7AU

References

KARLOWSKI, T. R., CHALMERS, T. C., FRENKEL, L. D *et al.* (1975) Ascorbic acid for the common cold. *Journal of the American Medical Association*, **231**, 1038–1042

KRAMER, M. S. & SHAPIRO, S. H. (1984) Scientific challenges in the application of randomised trials. *Journal of the American Medical Association*, **252**, 2739–2745

MARKS, I. M., LELLIOTT, P., BASOGLU, M., *et al.* (1988) Clomipramine, self-exposure and therapist-aided exposure for obsessive–compulsive rituals. *British Journal of Psychiatry*, **152**, 522–534.

NEWCOMBE, R. G. (1988) Evaluation of treatment effectiveness in psychiatric research. *British Journal of Psychiatry*, **152**, 696–697.

OXTOBY, A., JONES, A. & ROBINSON, M. (1989) Is your 'double-blind' design truly double-blind? *British Journal of Psychiatry*, **155**, 700–701.

Reviewing reviewers

SIR: I am writing to defend Macdiarmid's review of Ellenberger's *The Discovery of the Unconscious* (*Journal*, January 1990, **156**, 135–139) against what I consider to be a naïve and rather arrogant criticism by Brooks (*Journal*, May 1990, **156**, 747).

I would consider that skimming, followed by selective 'dipping', is an important part of the reviewers art. This may indeed proceed to more thorough reading and re-reading, depending on the nature and merits of the text being considered. Macdiarmid himself clearly appreciated this long book as a reference text and for selective re-reading. In this he was realistic as well as admirably honest.

I take issue with the suggestion that reviewers must read their material word for word. Indeed, the logical extension of such an opinion is that an adequate review of the *Oxford Dictionary* would require a reincarnation of Samuel Johnson. Pedantry leaves little room for intuitive judgement. Indeed, little academic work of any sort would be accomplished if writers felt it incumbent on them to read every word of the literature in the area of their interest, or to know all about a subject before feeling free to comment. Such attitudes inhibit the creative enterprise. Psychotherapists who work with creative people know this.

Additionally, I suspect that Dr Brooks may himself have committed the crime of which he accuses Macdiarmid. He suggests in his letter that Macdiarmid fails to take note of the proportion of the book given over to Janet, and its emphasis on the importance of the work that came before Freud and influenced his thinking. For me this came over clearly in the review.

I liked Macdiarmid as a writer. He showed respect for an important and scholarly book. In particular I liked his elegant and witty 'attack-from-within' on some of the more pretentious aspects of the psychotherapeutic establishment. On reading Macdiarmid I return with renewed appreciation to Ellenberger. Surely this was an excellent review!

C. A. COGHLAN

Ealing Hospital
Uxbridge Road
Southall
Middlesex UB1 3EV

ROC analysis

SIR: I hesitate to take issue with Snaith & Owens' (*Journal* May 1990, **156**, 744–745) recommendations about presenting the results of a relative (or receiver) operating characteristic (ROC) analysis. ROC analysis examines the ability of a screening instrument to discriminate cases and non-cases across the whole spectrum of morbidity by plotting sensitivity against false positive rate for all possible cut-off points. However, there are good reasons for displaying a smoothed ROC curve rather than "a series of straight lines joining the points" as they suggest.

Unless the sample size is very large in relation to the number of scoring categories, the selection of a cut-off point from the *actual* as opposed to *estimated* data can be very misleading. Random bunching of response scores can result in apparently excellent results (in terms of sensitivity, specificity and overall misclassification rate) with a chosen cut-off point, whereas a slight alteration in the cut-off produces a much poorer result.

The best way to avoid giving a false impression is to show the smoothed ROC curve with 95% confidence intervals on either side. A convenient computer program (ROCFIT) is available which calculates the maximum-likelihood fitted ROC curve and other parameters (Metz *et al*, 1984).

The optimal cut-off point (that is, the best trade-off between sensitivity and specificity) is at the point on the ROC curve which is the greatest perpendicular distance from the diagonal. Of course, *any* cut-off point may be chosen for a particular purpose: the smoothed ROC curve will give a maximum-likelihood estimate of the resulting sensitivity and specificity.

DAVID B. MUMFORD

Department of Psychiatry
University of Leeds
15 Hyde Terrace
Leeds LS2 9LT

Reference

METZ, C E., WANG, P-L & KRONMAN, H B (1984) ROCFIT Chicago: Department of Radiology and the Franklin McLean Memorial Research Institute, University of Chicago

The Lomax affair

SIR: I read Harding's reappraisal of Lomax's contribution with great interest (*Journal,* February 1990, **156**, 180–187). However, lest the impression be gained that asylums were places of brutality and inhumanity in general, it should be pointed that Lomax described conditions as he found them between 1917 and 1919 – a period when conditions were highly unusual. Not only had many younger medical staff been called up, leaving men past retirement age to manage alone, but many attendants too had gone to war, leaving the asylums grossly under-staffed. Nor was that all; some asylums had been taken over for war casualties and thus other asylums (presumably including Bracebridge and Prestwich) became grossly overcrowded.

There were also severe shortages of food – a matter beyond the control of the asylum managers. In the Burntwood asylum, for instance, the meat allowance had been reduced from 1 kg per head weekly in 1916 to 0·64 kg in 1918; heating also was almost certainly inadequate as the cost of coal escalated. During 1914 there had been 108 deaths within the asylum, but in 1918 there were 256, and these figures cannot be explained by an outbreak of influenza which accounted for only a few deaths. The strong implication is that malnutrition occurred. Nor was the Burntwood asylum unique: in the Worcestershire asylum there were 134 deaths during the final quarter of 1917, whereas during the whole of 1916 there had been 148 deaths.

Thus, if Lomax found "... poor nutrition ... and a high death rate ...", then this finding is in harmony with what was happening elsewhere and is a reflection of the harsh conditions which prevailed at that time. Of course, there can be no excuse for brutality and perhaps Harding's comment that "Prestwich was undoubtedly a badly administered asylum" is justified. My own reading of the history of the Burntwood asylum (Budden, 1989) has revealed nothing like that; on the contrary, there were summary dismissals of staff at the slightest suggestion of ill-treatment of patients, whatever the provocation.

No doubt Lomax was well justified in describing the conditions he found and in seeking improvements. However, it would be grossly unfair to use the description of conditions during 1917–1919 to castigate the asylum service and to denounce the provision which had been made by the Victorians for their mentally afflicted citizens.

D. W. BUDDEN

Pharmacy Department
St Matthew's Hospital
Burntwood
Walsall
West Midlands WS7 9ES

Reference

BUDDEN, D. (1989) *A County Lunatic Asylum. The History of St Matthew's Hospital.* St Matthew's Hospital, Burntwood: D Budden.

Brain transplants: myth or monster?

SIR: Henderson (*Journal*, May 1990, **156**, 645–653) cites brain transplants (in brackets) as one of the possible future treatments for dementia. The question which has been left unanswered is: who will be the donor? Will Mr A receive Mr B's (or Mrs B's) brain tissue with which to think and control his own body, or will B (male or female) wake up (again!) in A's body?

I put this question informally to a number of psychiatrists and got almost as many different replies. What I did learn was that the debate between Descartes and Franz Alexander was alive and kicking (Brown, 1985). Some psychiatrists see the mind as being synonymous with the physical brain, others view it as some ethereal envelope about our person, whereas still others conceptualise it in terms of a supernatural and eternal soul which utilises an otherwise selfless brain during life.

Will brain transplantation join the growing list of procedures requiring lengthy ethical (and theological) debates along with predictive genetic testing and organ donation from anencephalics? Or, are the technical aspects of full brain transplantation so complicated as to render the whole argument academic or fictional? Perhaps partial brain transplants would not arouse the same emotions, but which parts and how much?

BRIAN O'SHEA

Newcastle Hospital
Greystones
Co. Wicklow
Ireland

Reference

BROWN, T. M (1985) Descartes, dualism and psychosomatic medicine. In *The Anatomy of Madness, Vol. I* (eds W. F Bynum, R. Porter & M. Shepherd), pp. 40–62 London: Tavistock

Patterns of attendance at child psychiatry clinics

SIR: I read with interest the paper by Stern *et al* (*Journal*, March 1990, **156**, 384–387) and would like to offer local experiences in Hong Kong to widen the discussion.

In a seven-year survey of referrals to the child unit of a department of psychiatry in a teaching hospital in Hong Kong (Luk & Lieh-Mak 1985), somatic complaints were the most common reason for referral (19.6%). When we opened a self-referred primary mental-health care clinic for children at a well-established voluntary agency, conduct problems became the most common complaint (46.4%; Chung & Luk, 1990). The clinical diagnoses made also differ between the two settings. While these might be explained by referral bias, Hong Kong parents did seem to seek help for different problems from different sources: somatic problems from doctors, and 'bad' behaviour from non-hospital settings. The fact that Drs Stern *et al* found Asian children referred had a narrower range of problems might be explained in this way. Furthermore, when the primary mental-health care clinic for children was set up in Hong Kong with minimal advertisement, a half-year waiting-list promptly accumulated: Hong Kong parents, acutely aware of their 'problematic child', actively sought help, but unfortunately not usually from hospital. This might shed light on why Asians were under-represented in Dr Stern *et al*'s paper. Although Dr Stern *et al* did point out that the department accepted self-referrals, it was the hospital setting itself that carried the stigma. Those parents who think that their child has problems might not know or like the idea of attending child psychiatric clinics in hospital settings. The under-representation of Asian children can therefore be explained not only in terms of cultural differences concerning what is acceptable behaviour in children, but also in terms of the parents' perceptions of who and what problems should be taken to a child psychiatric clinic in a hospital.

Ting-pong Ho

Department of Psychiatry
Queen Mary Hospital
Hong Kong

References

Chung, S Y & Luk, S. L. (1990) Characteristics of children attending a primary care psychiatric clinic *Journal of Child Psychology and Psychiatry* (in press)

Luk, S. L. & Lieh-Mak, F. (1985) Child psychiatry in Hong Kong. *Journal of Paediatrics, Obstetrics and Gynaecology*, 15–18.

First-rank symptoms

Sir: O'Grady's review of first-rank symptoms is most welcome (*Journal*, April 1990, **156**, 496–500). It is a pity, however, that his summary chose to highlight the newsworthy rather than the useful aspect of his study.

Using the normal definitions, all 13 first-rank positive patients in his sample had Research Diagnostic Criteria (RDC) schizophrenia or schizoaffective disorder. Indeed, almost three-quarters of his RDC schizophrenics showed first-rank symptoms. This makes Schneider's symptoms very useful as a diagnostic aid.

'Widening' (i.e. blurring the definitions) reduced their selectivity without increasing their sensitivity.

A. C. Carr

Department of Psychiatry
Hamilton Civic Hospitals
237 Barton Street
Hamilton L8L 2X2

Sir: Trimble (*British Journal of Psychiatry*, February 1990, **156**, 195–200) manages to side-step the problematic concept of schizophrenia by proposing a psychopathological cause of the first-rank symptoms. Thus he brings psychiatry closer to neurology, and increases our hopes that, just as a particular abnormality of the expression of speech may be localised to a particular area of the brain, so, one day, we might find that a particular symptom of schizophrenia is associated with a discrete cluster of abnormal neurones. Such an approach also allows us to accommodate, without any difficulty, the similarities (Johnstone *et al*, 1988) and differences (Cutting, 1987) in phenomenology between different psychoses. The clock is turned back from Schneider to Bleuler since these psychotic symptoms can, once again, be given a central psychopathological role.

However, although Dr Trimble convincingly argues that the first-rank symptoms occurring in temporal lobe epilepsy arise in the temporal lobe, he then suggests that *all* such symptoms arise in the temporal lobe ("If we are to regard first-rank symptoms as specific to the temporal lobe, then we must say that they all arise there"). The evidence for this is, firstly, that schizophrenia is associated with temporal lobe abnormalities and, secondly, that the temporal lobe is ideally placed to produce all the first-rank symptoms. Indeed it is, but then it is also ideally placed to produce all other disorders of thought and perception. We are left with the conclusion that first-rank symptoms arise in the temporal lobe, but might also arise elsewhere, and that temporal lobe pathology causes first-rank symptoms, but is likely to cause other disorders of thought and perception. Thus, these symptoms seem rather *non*-specific.

Furthermore, the first-rank symptoms, as with all psychotic beliefs and hallucinations, are unlikely to each be unitary phenomenon (i.e. even these symptoms are themselves 'clusters' of symptoms). It is not just the experience but the interpretation of that experience that justifies the label of 'psychotic'. In other words, each first-rank symptom can itself be subdivided into the 'experience' and the cognitive appraisal of that experience (which may or may not be accurate), although these events may be experienced as simultaneous and therefore as a unitary phenomenon. Dr Trimble's theory is unable to elucidate which of these different components arise in the temporal lobe.

First-rank symptoms are complex phenomena that arise only if there is a failure of information processing in a number of different areas of the brain. We lack both a coherent understanding of the mechanisms underlying the production of psychotic phenomenology, and knowledge of the many pathways whereby these phenomena may be produced. Like the butterfly of psyche, they continue to evade our attempts to pin them down to a specific locality of the brain.

Frances Klemperer

Guy's Hospital
London SE1 9RT

References

Cutting, J. (1987) The phenomenology of acute organic psychosis *British Journal of Psychiatry*, **151**, 324–332

Johnstone, E. C *et al* (1988) Phenomenology of organic and functional psychoses and the overlap between them *British Journal of Psychiatry*, **153**, 770–776

British Journal of Psychiatry (1990), **157**, 304–306

Books Reconsidered

The Theory and Practice of Group Psychotherapy: Irvin D. Yalom*

This book first appeared in 1970 and has gone into two further editions, one in 1975 and this one in 1985. Yalom is also the author of *Existential Psychotherapy* (1980), *In-patient Group Psychotherapy* (1983), the co-author with Lieberman of *Encounter Groups: First Facts* (1973) and with Elkin of *Every Day Gets a Little Closer: A Twice-Told Therapy* (1974) (which recounts the course of therapy from the patient's and the therapist's viewpoint). The present book is the central work of the set and seems to me the most substantial. It is also one of the most readable of his works because of its straightforward style and the liberal use of clinical examples.

The book's first appearance followed the burgeoning of interest in all types of group treatment in the 1960s, when it seemed to some, especially in southern California, that groups could achieve anything. When I trained in the 1970s, group therapy was definitely 'in' and Yalom was the guru. During the 1980s there was a swing away from all that. Advances in biological psychiatry and the considerable improvements in the understanding of behavioural psychotherapies, arising from solid research effort, have diverted elsewhere the interest of young psychiatrists. Within the psychotherapy field itself there is perhaps also a strengthening of theoretical approaches other than Yalom's.

A virtue of Yalom's book is that it brings to the written page some of the infectious excitement of its age. At the same time, it is a scholarly work in which research is critically reviewed and its lessons incorporated into guidance about clinical practice. In this respect, Yalom is one of the very best writers in the psychotherapy field. He recognises that we are apt to deceive ourselves about the effects of what we do and that systematic research may be the only valid way of avoiding this. He points to those areas which are yet little explored by research; but he is also a careful and thoughtful clinician whose teaching is based on the many hours he has spent in groups of his own and on the observations of the many other therapists he has supervised. This balance between detailed guidance from clinical experience and an analysis of the clinical lessons to be learned from the research literature is rare in psychotherapy writing which seems, on the whole, not to be informed by research results – in contrast to the literature about behavioural therapies.

Many readers of this Journal will be familiar, as I am, with the second edition of this book. For the third edition Yalom has incorporated both new knowledge (such as work on pre-therapy preparation), changes in fashion (such as the relative decline in encounter groups) and clearer guidance about the running of specialised groups (such as groups of in-patients). He has done this without lengthening the work significantly and without losing important material.

The book begins by raising the question of how group therapy helps patients to change, and introduces the concept of the 'therapeutic factors'. Yalom bows to recent usage by dropping his earlier use of 'curative factors'. There are certainly indications that these factors may help group members, but the term 'curative' claims too much. Yalom spends four chapters looking at the nature of each therapeutic factor and how it might operate in groups. He summarises most of the relevant research, and provides a particularly good introduction to thinking about how group therapy might help its participants.

Yalom could outline the historical development of the concept of therapeutic factors more fully, however. Essentially, the concept comes from a sampling of clinicians' ideas about what is therapeutic in groups. Different authors have developed their own lists of factors, although these are usually based on that by Corsini & Rosenberg (1956). It is difficult to know whether these lists exhaustively describe the therapeutic elements in an economical way that contains no overlap. My impression is that Yalom himself irritates some therapists by not listing 'working through transference(s)', although his later chapter dealing with "Transference and transparency" clearly shows that he regards transference as a very important element of therapy. A historical perspective would do more to show why this is so and why transference elements of therapy are mostly subsumed under 'interpersonal learning'.

---

*New York: Basic Books (1985) (3rd edn)

Yalom describes how therapeutic factors may lead to change, and discusses the relative importance of the different factors. He puts most emphasis on interpersonal learning and group cohesiveness. For Yalom the group is a 'social microcosm' in which the patient can experience relationships with a variety of types of people (hence the value of heterogeneity discussed later) including an authority figure (the therapist), express feelings about these relationships, receive feedback that may reveal how his or her perceptions contain distortions from reality, and practise new ways of behaving and relating which can later be used in the real world. Thus, interpersonal learning contains embedded within it elements of transference resolution, the gaining of insight and even graded practice. Clearly the group needs to be a safe and comradely place for the individual to feel free to experiment in this way. In the cohesive group, the patient feels accepted 'warts and all' and the esteem of his or her fellow patients may be the basis for increasing self-esteem.

Yalom moves on to deal with the responsibilities and tasks of the therapist in conducting therapy groups. The therapist creates and maintains the group, builds the group culture and sets norms of behaviour. Yalom emphasises the importance for the therapist of working in the 'here and now'. This is a reflection of his major focus on interpersonal learning. Here, as elsewhere in the book, he gives many relevant clinical examples taken from his own practice. The therapist should use the patient's past experience as a tool for illuminating the present; the 'archaeological exploration of the past' has no place in the group.

Yalom discusses the therapist's responsibility for removing obstacles to progress that may arise and he/she may do this by using group-process commentary. Novice therapists often feel bewildered about process interpretation – experiencing either a confusing welter of contradictory ideas or a complete blank. Yalom provides excellent guidance by emphasising the function of interpretation in relation to patients' presenting difficulties.

He discusses the work of Bion and what he refers to as the 'Tavistock approach', where the emphasis is on 'group as a whole' interpretations. He points to outcome research which suggests that 'group as a whole' interpretations are weak factors in producing change in individuals and that their sole use leads to ineffectual group therapy.

Chapter seven, entitled "The therapist: transference and transparency" contains some of the most valuable material in the book. Certainly the beginning therapist will find the discussion of transparency of immense practical value. Yalom observes that transference is a powerful aspect of the group's reality, that the therapist must recognise and utilise aspects of transference and that there must be resolution of the distorted attitudinal set towards the leader as part of the work of the group. He discusses these issues in detail, but he parts company from the orthodox group-analytic line (if such exists), and makes it clear that the analysis of transference is not the major task of the group. Here Yalom shows his debt to Sullivan and his interpersonal theories, a debt which he acknowledges earlier in the book. In these views Yalom is by no means alone and there are other writers, such as Whitaker and Lieberman (whom Yalom cites), who have developed similar ideas, although expressed in different terms (a familiar bugbear of psychotherapy writing).

My own feeling here is that Yalom could easily go a little further and build a bridge between the psychoanalytically-based psychotherapies and the newer cognitive-behavioural therapies that are emerging from the behavioural tradition. The interactional focus of his work and the emphasis on interpersonal learning betray many of the same concerns that are addressed in cognitive-behavioural therapies. There would be value in tracing the similarities between these therapies which have such diverse derivations, as well as clarifying what is different.

Transparency, or the degree to which the therapist reveals himself, is held to be of great importance. To Yalom, the analytic 'blank screen' is not a productive element of group therapy. It is an appropriate degree of transparency that promotes resolution of the transference. The patient can check his projections onto the therapist with the therapist's actual reactions. A wide-ranging discussion here brings in both research results and references to Eugene O'Neill and Hendrik Ibsen in order to illustrate both the advantages and the drawbacks of transparency and to help define its limits.

The three following chapters deal with preparatory elements of running a group – selection of patients, group composition, preparation of patients for therapy and the setting for group sessions. Selection for therapy is dependent on the aims of the particular group, and the research evidence relating to selection is scanty. Therefore Yalom pulls together the consensus of clinical opinion on the topic and draws on his own clinical experience. He reports some of his own work on dropouts from group therapy which throws light on reasons for excluding some patients from out-patient therapy groups, but there is little to learn from research about inclusion criteria. Intensive heterogeneous out-patient groups should exclude brain-damaged, paranoid, hypochondriacal, addicted, sociopathic and acutely psychotic patients, although there is evidence that specialised and more homogeneous groups may produce benefits for patients in some of these categories.

He introduces his chapter on group composition with a section on predicting how patients will behave in therapy. Various types of intake interview procedure have attempted to make this prediction and Yalom's conclusion is that the more the intake procedure mimics the group situation itself then the more accurate will be its predictions. From clinical observation there seems to be a good deal of advantage in heterogeneity in group composition, at least for intensive out-patient group therapy. That is, the patients should not all be too alike in terms of problem type and areas of psychological conflict, although the heterogeneity needs to be within a fairly narrow range so that scapegoating does not occur.

The chapter on creating the group gives advice on the physical setting of the group and its boundaries in terms of duration, frequency of meetings and group size. A section new to this edition deals with the controversy over whether patients should have preparatory sessions for group therapy or whether the group should be a mysterious place with an enigmatic, opaque therapist where the patient will rapidly develop transference to the therapist and to other patients. Yalom is clearly in favour of preparation on the basis of a fair amount of research evidence that suggests it enhances the work of groups.

There is a section on the marathon group or time-extended therapy session, proposed as a way of focusing and enhancing the work done in regular sessions. As Yalom states, this is a fashion that has come and gone. Perhaps he will drop this section from the next edition, but he leaves it in here as a way of illustrating the passing fads that affect group therapy, and he introduces his own research which throws doubt on the effectiveness of marathon sessions.

Two chapters deal with the evolution of the group from the first meeting to termination, setting out some of the events and stages of development that are commonly seen in groups, together with common developmental problems such as subgrouping. There is a section on membership problems, dropping out, adding new members – and how to throw out an unsuitable member! Naturally enough these chapters end with a section on termination.

Chapter 13 deals with particular types of patient liable to cause difficulties in out-patient therapy groups. Some of these are presented in diagnostic terms (e.g. the schizoid patient, the borderline patient) and others in behavioural terms (the silent patient, the self-righteous moralist). Clinical examples are given illustrating how these difficulties may be overcome in a manner which has a chance of leading to therapeutic gain.

Chapter 14 is where Yalom writes about a number of topics he cannot fit in anywhere else. However, these are all important issues which are discussed among trainee therapists: concurrent individual and group therapy, the value of co-therapy, the leaderless group, the use of dreams, videotape feedback, written summaries and structured exercises (e.g. 'warm-up' procedures).

Chapters 15 and 16 deal with other types of group than the intensive out-patient therapy group and are in some ways condensed versions of two of his other books *In-Patient Group Therapy* and *Encounter Groups: First Facts*. Group work has been a popular feature of acute in-patient units in recent years and is often applied without clear goals, nor is there research evidence of its value. Thus Yalom's thoughtful analysis is very helpful. Encounter groups, on the other hand, have almost passed into history, but elements of the movement have transferred themselves into other fields, such as religious organisations, skills training workshops and self-help groups.

Yalom rounds off the book with a chapter on the training of group therapists and a comprehensive chapter-by-chapter bibliography.

This book was born 20 years ago, years that have seen great advances in other psychological therapies and changes of fashion on the group therapy scene. Nevertheless, the uniqueness of the book remains in providing a comprehensive introduction to group psychotherapy that spans both clinical experience and research results. Both the trainee who wants an idea of the nature of group therapy and the colleague who needs an introductory manual to supplement supervision in running his or her first group should read the work right through; for the academic interested in group therapy research and for the experienced group therapist it should be readily available on the shelf to dip into.

E. C. Crouch, *Consultant Psychotherapist, St John's Hospital, Stone, Aylesbury, Bucks HP17 8PP*

British Journal of Psychiatry (1990), **157**, 307–317

Book Reviews

Editor: Sidney Crown

Cognitive Therapy for Depression and Anxiety. By IVY-MARIE BLACKBURN and KATE DAVIDSON. Oxford: Blackwell Scientific. 1990. 230 pp. £19.50.

This book provides a clearly written account of Beck's cognitive therapy by two authors who have contributed to its development and evaluation in the UK. It offers probably the most accessible description of the approach and would enable any moderately experienced professional to learn its main techniques; general psychiatrists who lack specific psychotherapeutic skills would also be well advised to read it. The basic model is a simple or simplistic one: depression and anxiety, which are the main topics of the book, are caused by our schemata (i.e. stable knowledge structures) and by our faulty information processing (selective abstraction, arbitrary inference, over-generalisation, magnification and minimisation, and personalisation) which lead to faulty inferences and consequently to negative mood states. Therapy resembles a structured teaching programme in which personality is largely, and historical data entirely, ignored; the therapist suggests hypotheses, sets structured tasks and challenges beliefs with the aim of modifying the faulty cognitive processes.

The last sentence of the book reads "... cognitive theories provide one level of understanding the emotional disorders and one pragmatic approach for treatment which do not preclude other levels of understanding or other aetiological theories." The sophisticated reader will be well advised to read this paragraph first, for the general tone is one of ineffably confident common sense and, despite the extensive reference lists, little or no attention is paid to the limitations of the approach or to its relation to other theories and methods. The "easy-to-use, modern page layout" and the washable cover add to the sense of being condescended to. The sophisticated patient may also find cognitive-therapy techniques a little facile and the problem of non-cooperation in therapeutic work, whether from sophisticated or unsophisticated patients, is hardly addressed at all. Clearly a proportion of patients will respond well to a pure cognitive therapy approach, although little guidance is given as to how to recognise them, but the techniques can have a much wider application as one part of a more general and, I would urge, dynamic psychotherapeutic approach.

A. RYLE, *Consultant Psychotherapist, Department of Psychotherapy, St Thomas's Hospital, London*

Depression in Schizophrenia. Edited by RICHARD WILLIAMS and J. THOMAS DALBY. New York: Plenum Publishing. 1989. 271 pp. $62.50.

This book, based on the proceedings of the World Psychiatric Association symposium in Calgary (1988), covers issues of theoretical and practical importance which are much debated but still largely unresolved. Although the stated intention of the symposium was to "draw together leading international researchers", the 36 contributors were exclusively from centres in North America and the UK. Within this limitation, however, the range of expertise was impressive and comprehensive, with papers falling into four sections: theoretical aspects, phenomenology, suicide and prognosis, and treatment approaches. The chairmen of each of these sections give brief introductions to their relevant chapters, but there is no reference to any subsequent discussion (assuming that there was some) and rather limited attempts at drawing together and evaluating the often conflicting strands of evidence, although this is admittedly difficult and a problem also for the reviewer!

"Theoretical aspects" deals with the thorniest issues. What is schizophrenia? What is depression? Do they even exist? What about schizoaffective disorder? The concept of schizophrenia as a diagnostic entity is rejected by Brockington who postulates a complex three-dimensional descriptive approach, while McGuffin *et al*, in a review of the contribution of genetics, highlight the generally poor concordance in the diagnosis of schizophrenia using the different diagnostic criteria currently available, despite the fact that these are all highly reliable individually.

The eight chapters which address the issue of phenomenology of depression in schizophrenia again attempt to tackle some old and largely unanswered questions. Is the depression a reaction, a concomitant or a result of antipsychotic medication? Is it really depression anyway or rather the negative symptoms of schizophrenia? The conclusion must be that we still do not know but that new approaches, techniques and measures should hopefully help in finding the answers.

Three papers deal with the long-recognised association between suicide and schizophrenia, but the section chairman, Tanny, considering three widely divergent explanations for the linkage, concludes that the choice between them remains unresolved.

The last, and most immediately practical, section is concerned with treatment approaches and necessarily reflects the underlying conceptual complexities and uncertainties addressed in the previous papers.

If there were no clear conclusions at the end of the conference, the book nevertheless presents a thorough overview of current thinking in the area and should stimulate and guide further work. It deserves to be read by a wide audience.

Michael Pritchard, *Reader, Department of Psychiatry, Guy's and St Thomas's Hospitals, London*

Anxiety and Stress Management. By Trevor J. Powell and Simon J. Enright. London· Routledge. 1990. 196 pp. £9.99.

This book aims to provide a practical guide for health workers treating anxiety and stress, although the authors also hope it will be useful for patients. The book is primarily cognitive-behavioural in approach, and includes chapters on models of stress, classifications and theories of anxiety, assessment, anxiety management techniques, running anxiety management groups, and individual case examples Unfortunately, this wide range of topics means that the coverage of each topic is correspondingly thin. The general impression is of a superficial survey of many different approaches, but with little sense of an integrated model which could be flexibly applied in clinical practice.

The writing is generally clear and down to earth, but the range and choice of topics is sometimes odd. The chapter on classification and theory contains lengthy descriptions of DSM–III criteria for anxiety disorders and sections on psychoanalytic theory and neurophysiology, none of which seem very relevant to the relatively naive therapist seeking practical guidance, or to patients. The lack of depth is felt particularly when the book turns to treatment. Most of the techniques described, which should surely be the core of a 'practical guide', are covered in one fairly short chapter, which rushes through such diverse approaches as relaxation, controlled breathing, distraction and cognitive therapy (three different schools of which are covered in six pages). I was left unsure about who would find this helpful. The inexperienced reader (at whom it seems to be aimed) is unlikely to be able to make good use of techniques so briefly described, while more experienced readers will learn nothing new.

Some chapters, such as those on models of stress and on assessment, do provide reasonable introductory summaries for the newcomer, and some patients might find parts of it useful, but, in general, the attempt to reach both therapists and patients leads to the book missing both targets.

David Westbrook, *Senior Clinical Psychologist, Warneford Hospital, Oxford*

Psychological Factors in Competitive Sport. By Don Davies. Basingstoke: Falmer Press. 1989. 177 pp. £18.95 (hb), £7.95 (pb).

If health psychology is the flavour of the month, sports' psychology is a much publicised component. While no one who has played a game seriously would doubt the importance of psychological factors, the problem is what factors or combination are effective for a particular person in a particular sport.

This book applies to sport in a readable, although somewhat dogmatic way, a number of principles from the psychology of learning, motivation, skill, practice, feedback, anxiety and stress, concentration and confidence generation. Useful enough, no doubt, and a great deal better than nothing, but the book gives a clear impression of an applied science in its infancy much in need, to judge from the dates of many of the references, of up-to-date research.

Most of the examples are drawn from tennis, and it is difficult to accept the implicit argument that the same principles apply in more than a general way to, say, mountain climbing, skiing, hang gliding, fishing or snooker. For anyone wanting a sports' psychology book for themselves or to recommend to patients Syer & Connolly's *Sporting Body, Sporting Mind* (Cambridge University Press, 1984) is better: less pretentious, better presented and with a background of a variety of sports.

Sidney Crown, *Consulting Psychiatrist, The London Hospital, E1*

Brief Therapy Approaches to Treating Anxiety and Depression. By Michael D. Yapko. New York: Brunner/Mazel. 1989. 384 pp. $39.95.

This book is based on the fourth conference in hypnotic and strategic interventions held at the Milton H. Erickson Institute in San Diego, California in 1988, although it does not read in the disjointed way which abstracts from a conference often do. It becomes clear that this is a collection of papers almost exclusively by those at the Erickson Institute, and as such reflects the rather narrow focus of a style and viewpoint based on the use of hypnotic techniques within an active, very positively connotated style of therapy. Reading the book largely at one sitting, the relentlessly cheerful style had rather a depressing effect on this reader!

The book consists of an entertaining keynote address by Seligman (based on his work on attributional style and depression), followed by a long section entitled "Intervention for depression" and a shorter one "Intervention for anxiety", and ending with a short section entitled "Therapeutic approaches for anxiety and depression". The best features of the book are that it is liberally interwoven with clinical vignettes, although some of these were too schematic, and that the creative use of story and metaphor, especially where resistance was a problem, was interestingly developed, particularly in the chapter on multisensory metaphors for helping children with fear and depression (chapter 12).

Throughout, familiarity with the methods and writings of Milton Erickson was assumed. There was overduplication of categorisations of depression as well as repeated descriptions of such techniques as reframing, some judicious editing would have led to a slimmer, more digestible volume.

Schwarz's chapter on the treatment of anxiety and depression in pain states was thought-provoking and original, as was the short chapter entitled "When doesn't the problem happen?" which suggests ways of using the patient's inner resources to resolve their problems. At times, however, the writing sank into a laboured style: for example, on p. 184 depression is described as "a multifaceted phenomenon that stands as one of the most common psychobiological maladies." Throughout, the emphasis is on a solution-orientated approach to problems, with an active therapist and client. It is worth dipping into this book, and the reader may well enjoy some of the pithy proverbs quoted, for example, "When the vine entwines your roof, it is time to cut it down" (p. 97). In the end, however, I was left with a feeling of having had a surfeit of southern Californian hedonism; the readers must judge for themselves.

TERESA BLACK, *Department of Psychotherapy, Manchester Royal Infirmary, Manchester*

The Suspended Revolution. Psychiatry and Psychotherapy Re-examined. By DAVID HEALY. London: Faber & Faber. 1990. 233 pp. £14.99.

There has been a recent increase in writing about phenomenology and psychopathology, as if there is a growing feeling in psychiatry of needing to go back to basics in order to make fresh developments. The author of this book argues that psychopathology should regain the central place in psychiatry that it has lost and which has led, in his view, to a fragmentation of knowledge and a stalemate between organic and psychological models in explaining and treating psychiatric conditions.

He gives a brief historical review tracing the development of ideas about brain, mind and psyche and the confusions that have arisen by the changing usage of common terms. He uses the concept of organisational levels to differentiate brain, mind and psyche, but also the links between them, so that the psyche is a psychological system arising from possibilities inherent in the nervous system but not predicted by them.

The remainder of the book looks at the confusion between these levels which is around in current thinking about the functional psychoses. His own hypothesis is that there is an underlying subtle biological disruption, which may be self-limiting, and that many of the symptoms associated with the psychoses (e.g. delusions, hallucinations) are psychological phenomena developed by the patient to explain their disturbing experiences, as was thought by Kraeplin, Bleuler and others. He develops ideas of how this occurs by looking at theories of illness behaviour and the confusion of reasons and causes leading to attributional errors.

If we followed the example of neuropsychologists and listened to what the patients told us, seeing them as the best investigators of their own condition, he argues that we could clarify these phenomena, rather than reifying them as diagnostic criteria as in DSM–III and getting caught in circular thinking that clarifies nothing

He points to recent advances in cognitive therapy which suggests that psychological treatments may be as powerful as organic ones, but that we are not using a theoretical framework that allows us to build on these findings.

Although written for a wide audience, I did not find this an easy book. However, it was enormously stimulating and should be read by all practitioners to rekindle interest in thorough descriptive accounts of the psychoses, leading to a more creative and focused way of applying treatment methods that make sense to the patient.

RACHEL LEHEUP, *Consultant Child and Adolescent Psychiatrist, Nottingham*

Working with the Person with Schizophrenia: The Treatment Alliance. By M. A. SELZER, T. B. SULLIVAN, M. CARSKY and K. G. TERKELSEN. New York: New York University Press. 1989. 399 pp.

This is a most unusual and welcome book. In it the authors describe their clinical approach to working with severely ill schizophrenic patients who are deluded, hallucinated or both. The book is rich in clinical detail, including transcripts of interviews demonstrating their method of holding patients in the treatment alliance, while at the same time, carrying out techniques which might be called reattribution. Effectively, these are the same age-old techniques used by Socrates.

The authors start from a psychotherapeutic standpoint but, in the introduction to the book, demonstrate their ideas of the biology of schizophrenia which are quite orthodox. They take strong, indeed passionate, issue with the view that the negative symptoms of schizophrenia are immutable and, in some patients, an inevitable part of the disease process. They do not deny a possible biological basis for some of these negative symptoms but their argument is that they are so difficult to identify and define and so similar, in some aspects, to voluntary behaviour that attempts must be made to treat them as an important part of the relationship between the patient and his/her carers.

I know of no other book which goes into such detail about the ways in which difficult schizophrenic patients can be helped to continue in a treatment alliance while, at the same time, having their delusional beliefs probed and challenged. Many sceptical readers might well say that there is nothing new in the book, that it simply details normal clinical practice. I doubt that this is the case. In the first place there are no other books which describe the experience in such detail (certainly not from modern psychiatry). In the second place, I doubt that many psychiatrists, particularly British ones, have the time or perhaps the inclination to spend regular 'psychotherapeutic' sessions with severely disabled schizophrenic patients who instead tend to be treated with much more behavioural paradigms in rehabilitation programmes. There are, therefore, lessons to be learned here for all of us.

Finally, one is struck with the immensely humanitarian approach of the authors Psychotherapy with schizophrenic patients has had a bad press since the rather punitive and psychedelic attempts of the 1960s. This book is a step in the rehabilitation of the psychotherapeutic approach to schizophrenia, modestly to rank alongside the new family therapists such as Leff and Falloon. In this individual approach the authors sensibly begun again at 'first base', the establishment of the treatment alliance. In all these respects, but particularly for eschewing more dogmatic psychodynamic approaches, the authors are to be congratulated.

CHRIS THOMPSON, *Professor of Psychiatry, University of Southampton*

Family Games: General Models of Psychotic Processes in the Family. By MARA SELVINI-PALAZZOLI, STEFANO CIRILLO, MATTEO SELVINI and ANNA MARIA SORRENTINO. London: H. Karnac Books. 1989. 285 pp. £14.95.

This book is as idiosyncratic in structure as it is in content, although its subject – families and family therapy – will be familiar to many readers. Co-authored by some of the original figures of the Milan School, many of the chapter titles, like the title of the book, offer little guidance to the real contents. Worse still, some of the terms used are idiosyncratic at best (e.g. imbroglio), and downright misleading at worst. For example, the authors point out in the preface that the commonest diagnosis of their 290 'psychotic' patients is anorexia nervosa! Yet, time and time again they refer to their patients as 'psychotic'. The term 'research' is used only in the psychoanalytic sense, a perspective otherwise long disavowed by this group of clinicians. The publishers inadvisedly repeat these problems on the back cover.

The early chapters acknowledge the limitations of the original Milan model, in particular its reductionism, and record how the original team increasingly diverged in their views on how change can be brought about in families. For *afficionados* of this approach the detailed anecdotal account by Selvini-Palazzoli and her colleagues will make fascinating reading, particularly the part concerning how the original prescriptive approach to therapeutic change developed into a much deeper appreciation of the complexity of human relations in families. For example, instead of their earlier and controversial disavowal of the importance of past history, the team now stress "the unforgoable need to bring history ... into all descriptions and explanations" (p. 262). For most readers, however, the book does not live up to its title. It is not about psychotic processes, nor is it really about families. Instead, the book is a detailed account of a number of families in therapy, recording the authors' deepening understanding of how change is brought about. It is not a book that can be easily dipped into, but only the most committed are likely to have the stamina to read the book from beginning to end. It is unlikely that it will provide help in introducing systemic ideas drawn from family therapy into general psychiatry practice.

ROBERT M. WRATE, *Consultant in Adolescent Psychiatry, Young People's Unit, Royal Edinburgh Hospital*

Handbook of Neurological Investigations in Children. By JOHN B. P. STEPHENSON and MARY D. KING. Guildford: Butterworth Scientific Ltd. 1989. 244 pp. £25.00.

This is a book about neurological investigations in children which does not contain formal descriptions of history taking and physical examination, although these are alluded to from time to time. It aims to be of use to "those who see children with suspected neurological disorders". Its potential readership could therefore include general practitioners and community medical officers, as well as paediatric neurologists, paediatricians and child and adolescent psychiatrists.

There are two main parts, the first concerned with investigations and the second with disorders or diseases. An appendix discusses the predictive value of investigation results and gives some normal values. The investigations section covers the major types, ranging from electroencephalography (EEG) to anti-epileptic drug monitoring. The investigations are described, followed by indications, contra-indications, and illustrative examples. The second section has chapters whose starting point is a particular presenting problem such as a floppy baby, peculiar gait, or regression. In these chapters, there is specific advice about the range of neurological investigations that are appropriate for a very wide range of conditions.

This book is packed with information. It is not a neurological text-book but it presupposes that readers are well informed about paediatric neurology. There is a very useful list of abbreviations in front of the introduction, but the use of technical terms emphasises the need for sophistication on the part of the reader.

There is an encouraging reference to Aicardi (1987) in the introduction which draws attention to laboratory investigations as only one source of data among those which need to be "integrated and shaped into a meaningful profile". That the section describing investigations precedes that describing disorders could be seen to run counter to this philosophy.

Exposition tends to be dogmatic, and although useful references are given at the end of the chapters, these are advice for further reading rather than specific justifications for statements in the text. The intent is to concentrate on practical advice to clinicians, and this means that investigations are only described in detail if they are considered to be in general use at the present time. In some cases, future use is not even referred to, as in the case of somatosensory evoked potentials in the prediction of cerebral palsy. In the second section, moreover, references to disorders can be rather telegrammatic.

There are other more surprising omissions such as the lack of explicit reference to Down's syndrome in the chapter on genetic investigations and to the investigation of neuropathic bladder in that on autonomic function. There is repeated evidence of the difficulty in deciding how much clinical paediatric neurology to include.

The authors are very aware that investigations occur in a context which includes the management of the child and their family. However, the broader psychosocial context is not addressed. It would be inappropriate for this book to discuss management in an extended fashion, but it would have been encouraging had there been a fuller reference to this aspect. In the section on disorders and diseases, there is economical 'no-nonsense' advice about relevant investigations.

In summary, this book is a very useful, although not fully comprehensive, source of information about neurological investigations which are currently available and the circumstances in which they are most appropriately used.

A. D. Cox, *Department of Child Psychiatry, Guy's Hospital, London*

Sexual Abuse. Incest Victims and Their Families. (2nd edn). Edited by Jean Goodwin. Chicago: Medical Publishers. 1989. 269 pp. £29.50.

This book is edited and largely written by Jean Goodwin, Professor of Psychiatry at Wisconsin, with contributions from other clinicians and researchers. As a second edition it contains added information on the adult victims of child sexual abuse and the longer-term sequelae including multiple personality disorder and post-traumatic stress syndrome.

The book is in three parts: evaluation and treatment planning, the sequelae of incest, and forensic and social implications of research in child sexual abuse. The book is well researched and good in parts. However, there is a tendency to be repetitive and idiosyncratic in choice of subjects. Whereas whole chapters are devoted to the incest pregnancy, the cinderella syndrome (simulated neglect), dissociative symptoms and hysterical seizures, there is virtually no mention of the links between sexual abuse and later mental illness and very little on possible social sequelae, such as links between victimisation and later victimising behaviour.

To read this book one would be forgiven in thinking that child sexual abuse was a phenomenon only affecting females. This is not the case and the particular problems of boy victims and later sequelae is not addressed. Similarly, the problem of extrafamilial sexual abuse is neglected. Although the book purports to deal with incest victims and their families, there is virtually nothing about the perpetrator of these offences, and little mention of family dynamics or of family therapy as a therapeutic tool.

Both the forensic section and the treatment planning section are disappointing and largely incomplete. Treatment issues are scattered throughout the book and as the word treatment does not figure anywhere in the index it is difficult to distil out the few pieces of information without reading through the whole book.

Nevertheless, this book may be of interest to mental health workers or social workers dealing with victims of sexual abuse but from an academic rather than a practical point of view. It draws attention to some of the more serious and unusual syndromes that can arise in survivors, but, apart from this, it adds little to the masses of information and new books that are accumulating on this subject.

Gillian Mezey, *Lecturer in Forensic Psychiatry, Institute of Psychiatry, London*

Current Approaches to the Prediction of Violence. Edited by David A. Brizer and Martha L. Crowner. Washington, DC: American Psychiatric Press. 1989. 171 pp. £22.50.

This book is one of the *Progress in Psychiatry* series which has emerged out of a co-operation between the American Psychiatric Association (APA) Scientific Programme Committee and the American Psychiatric Press. The series collates presentations from the symposia presented each year at the APA annual meetings.

The present book reviews biological, psychological, social and other multi-disciplinary approaches to the prediction of violent behaviour. The 15 US authors marshal evidence over 171 pages to support the conclusion that certain types of violence are predictable

The evidence is drawn from about 420 international references; most references are from between 1975 and 1985 although there are a few from earlier and later dates such as 1939 and 1988.

After the introduction, which presents an overview of current approaches for the prediction of violence, the nine different chapters deal with the following: models for the short-term prediction of violence potential; the prediction and cause of careers of criminal violence and assaultative behaviour in psychiatric in-patients; the clinical and historical correlates of dangerous in-patient behaviour; hierarchical neuronal regulation of aggression; environmental concomitance of psychiatric in-patient violence; child abuse; neglect and violent behaviour; and schizophrenic violence and psychopathology.

Most of the authors are well known outside the USA; they are all eminent in their respective field and have published widely within this particular sphere.

The editors have managed to mould this multi-authored book into a clearly understandable and succinct review. It is of some surprise though that no index whatsoever is included. Furthermore, more than 30% of the references of one chapter were by the author himself! More recent references, especially between 1986 and 1989, would have also added more value to this book.

Nevertheless, this book excels in the comprehensiveness of the topics it covers, and its conciseness. The summaries and notes at the end of some of the chapters, especially the chapter on short-term prediction of violence potential, are commendable.

A paperback version, less expensive and including an index, would have made this book invaluable for psychiatrists and other professionals working in the field of forensic mental health; it nevertheless should be available in all libraries offering advice for medico-legal questions.

H. Albrecht, *Specialist Psychiatrist, Forensic Services, Auckland, New Zealand*

Separation and the Very Young. By James and Joyce Robertson. London: Free Association Books. 1989. 256 pp. £12.95 (pb), £25.00 (hb).

Until relatively recently, the visiting of children in hospital was severely restricted. Relatives were excluded as possible sources of infection and as potential disturbers of ward routines. However, in the late 1940s and 1950s an appreciation of the distress suffered by young children who were left in hospital contributed to important changes in hospital practice, such as the provision of overnight accommodation. The work of James and Joyce Robertson, and especially James Robertson's films about the admission of young children to hospital, played an important part in catalysing these changes.

This book is a chronological history of the work of the Robertsons. It is a joint work that was finished just three weeks before James Robertson's death. The first part describes the making of the two hospital films and the reaction of the medical and nursing professions. In part two, their later work on children in foster care is described. Here, the emphasis is very much on the detailed observation of children's reactions to separation. The book concludes with a collection of papers on various aspects of child care, including adoption and custody and access.

I found this book a pleasure to read. As the introduction states, it is full of reminiscences, but it contains much that is relevant to practitioners working with children nowadays. The book provides a valuable social history of child-care practice in the past 40 years and deserves to be widely read.

Richard Harrington, *Consultant in Child and Adolescent Psychiatry, Birmingham University, Birmingham*

Our Responses to a Deadly Virus. The Group Analytic Approach. By Angela Molnos. London: Karnac Books. 1990. 240 pp. £12.50.

This book is written by a group analyst who has worked with carers involved in HIV/AIDS-related programmes. Her book addresses some of the difficulties they face in their work with people who are affected by HIV/ARC/AIDS.

In the first part of her book, the author highlights certain aspects of the AIDS epidemic, in particular, its effects on our language and attitudes. An example of AIDSpeak is the use of the term PWA for 'persons with AIDS' who are deeply offended if referred to as 'victims'. This lethal disease's association with homosexuality, sex and death threatens many of our social and moral values, creating a climate where prejudices and counter-prejudices can thrive.

It is by focusing on the social dimension of AIDS and its effects at the carer-patient interface that the author brings the reader to consider a group analytic approach in the care of people with HIV/AIDS. Our attitude to those we treat and to those who treat us will be intimately linked to our social expectations and even more so to the unconscious group fantasies and defences this interaction stirs up within us. These unconscious processes are clearly illustrated by the participants of a workshop on "Group responses to AIDS" which is the subject of the second part of the book.

As Cunningham says in her foreword, the third part of the book is "what the book is all about for those of us concerned with delivering effective health services". Through the use of staff support groups run on group analytic lines, Molnos stresses how we can become aware of unconscious processes which hamper our ability to cope and to work effectively. She illustrates this very clearly in her group work with health advisers treating people with HIV/AIDS. Not only do the participants confirm the value of her work but, perhaps more important these days, the reduced staff sickness, absence and turnover was seen to increase the cost-effectiveness of the service. This book is easy to read and clear, and, apart from details on how to run a workshop on group analytic lines, it is relevant to all who work in the field of HIV/AIDS and mental health.

FELICITY DE ZULUETA, *Consultant Psychotherapist, Charing Cross Hospital, London*

Abnormal Psychology: Its Experience and Behaviour. By PETER MCKELLAR. London: Routledge. 1989. 378 pp. £30.00 (hb), £12.95 (pb).

There is a distinct aura of the 1960s in this book which is interested in 'states of consciousness' and their relation to psychopathology and clinically abnormal states of mind. It has always been clear that orthodox scientific investigation is a poor tool with which to examine subjective experience, and McKellar therefore presents a largely anecdotal account, amounting to a travelogue across some weird and wonderful terrain. He also uses evidence from the field of art and literature, including the work of Dostoevsky, Coleridge, Dickens and Somerset Maughan, to illustrate his ideas. This is a refreshing and enjoyable approach, which highlights the aridity of much of the experimental literature. However, we cannot escape questions concerning proof and validity, and there are certainly reasons for concern on this score, particularly McKellar's use of evidence to make claims wider than those which can comfortably be based on it, as well as his dubious statements concerning the causation of abnormal states of mind.

The author sets out with two aims. The first is to understand abnormal states of mind as psychological phenomena, and the second is to investigate the relationship between these abnormal states and the subjective experiences of people we regard as normal. This emphasis on subjective experience leads to an attempt to account for complex emotions, defences and real-life experiences, and to the view that there are many subsystems of the personality, causing splits in consciousness which determine universal phenomena of mental life. McKellar illustrates these views through an examination of 'strange' phenomena such as dreams, hypnagogic imaginery, hypnotism, imaginery companions, *dejà-vu* experiences, synaesthesia and beliefs about spirit possession and the supernatural. He attempts to connect these phenomena with psychopathological states such as hysteria, personality dissociation, obsessions, phobias, schizophrenia, paranoia, depression and guilt. He views abnormal and normal states of mind as extremes on a continuum of experience, and he attempts to connect his principles with the wider psychological literature.

This book does not easily fit into the traditional genre of the psychology textbook, and one can imagine how it will be received by this particular academic community. This is both its strength and its downfall. It reminds us of phenomena which have been banished from thought or consideration within the academic community of psychologists (to others, it is obvious that subjectivity remains an important part of human mental life). However, this neglect has occurred with good reason since subjectivity is not readily investigated by strict scientific means. This causes some difficulty for McKellar, himself an academic psychologist, who attempts to use some of the tools of his trade in this book. The result is that there is a sense of things being disconnected: thought follows thought with no connecting theme. This means that there is no real cumulative argument and it is as if the author had not made sufficient sense of his material in his own mind before writing the book to enable the reader to understand it. I hope that McKellar's sense of dissatisfaction is sufficiently acute, and his curiosity strong enough for him to have another go at this neglected area, because it is sufficiently important for the results to hold considerable fascination.

TERRY BIRCHMORE, *Principal Clinical Psychologist, Dryburn Hospital, Durham*

Race and Culture in Psychiatry. By SUMAN FERNANDO. London: Tavistock/Routledge. 1989. 234 pp. £12.50.

There is little doubt about the multiracial status of the present British society. Since the Second World War, the arrival of more than a million immigrants from the New Commonwealth and Pakistan, together with their children born in the United Kingdom, has resulted in ethnic minority populations of more than two million people, and with them new forms of ethnic diversity. Psychiatry is a branch of medicine that had its roots in Europe and faces a challenge to accommodate race and culture within its clinical, diagnostic and treatment strategies. The present book is a useful addition to the long list of books published on the subject of transcultural psychiatry in the last decade or so.

Suman Fernando is a consultant psychiatrist, a mental health commissioner and chairperson of the Transcultural Psychiatry Society of the United Kingdom. He is more than an appropriate person to write a book on such a sensitive and important subject.

The book is divided into seven sections: the culture of psychiatry, current psychiatry, cross-cultural research, socio-cultural psychiatry, racist psychiatry, the race culture argument and, finally, a blueprint for change. All portions of the book are thoroughly researched with an excellent reference to the recent and the past psychiatric literature. The arguments are objective and supported by scientific research. The subject of race and culture in psychiatry is a vast one and it is a credit to the author to condense the subject into a very readable book of 200 pages. I found the first and second chapters informative, and the rest of the book depends on an understanding of the history of psychiatry, history of racism and resistance to change within psychiatry towards race and culture. The book attempts to dissect various aspects of psychiatry including research, publications, professional appointments and various bodies such as the Royal College of Psychiatrists in relation to race and culture.

Race and culture are sensitive subjects and many would like to deny their existence, especially in clinical psychiatry. This book is an eye-opener, however intimidating it might be to the reader. I recommend it to all professionals in psychiatry.

A. JAWAD SHEIKH, *Senior Psychiatric Registrar, University of Birmingham, All Saints Hospital, Birmingham*

Cults and the New Religious Movements: A Report to the American Psychiatric Association from the Committee on Psychiatry and Religion. Edited by MARC GALANTER. Washington: APA. 1989. 346 pp. £30.00.

This volume is the culmination of a three-year project designed to prepare a comprehensive report on the contemporary phenomenon of cults and new religious movements. It was undertaken by the Committee on Psychiatry and Religion and the American Psychiatric Association (APA) at the request of the Board of Trustees of the APA. It contains 16 major contributions from authors who tackle the problem from a variety of angles. While bearing in mind that the whole report is basically concerned with the North American scene, and some of the articles are concerned with North American law, it does present a very rounded view of some of the problems with which we are often faced in the UK. (The English situation is more fully, yet also more one-sidedly, expounded by Barker in *New Religious Movements*, HMSO, 1989.)

I would advise readers to begin with chapter 12 in which Richardson analyses the research methodology which has been adopted, the impact of the type of information gathered and the conclusions drawn from it. He points out that the researcher's perspective and discipline influence, if not dictate, the methods used to gather information. He demonstrates the variety of bias which accompanies research into such an emotive matter. This helps us to understand how many of the chapters arrive at almost opposite conclusions.

He also compares the cults with the process of psychotherapy, especially in regard to its non-specific elements. I found this a fascinating challenge when considering some of the new 'cults' of psychotherapy. Perhaps that is the next symposium which might attract the APA.

In chapter 16, Bromley & Shupe remind us of the fact that many of the present day conventional religious bodies have at one time been the object of varying degrees of controversy and violent opposition.

While I did not find this report very enlightening as to the individual cults themselves, I am sure that it would be a very important resource for those who may have to care for the parents of cult members or those cult members who have become disillusioned with their particular movement. It seems that many do so.

LOUIS MARTEAU, *The Dympna Centre, 60 Grove Road, London NW8*

Offending Women: Female Lawbreakers and the Criminal Justice System. By ANNE WORRAL. London: Routledge. 1989. 189 pp. £9.99.

The tone of this polemical book is set in the author's first paragraph where "the psychiatrist says she may suffer from epilepsy . . . and he may look into it." The author's outrage detracts from the book's message; as, for example, when she says that her book's aim is "to examine the ways in which authorisation of professionals and experts to define certain women as being the type of women who require treatment, management, control or punishment serves to perpetuate the oppression of all women."

Before moving onto the cases of 15 women "chosen (to) provide compelling illustrations of (or challenges to) my theoretical positions", one must wade through two tightly argued but impenetrable chapters which purport to define these positions.

The case descriptions are of some interest but occupy only 15 pages and relate to only 11 interviewed subjects. Regretfully, there are no full descriptions which enable one to ascertain whether these cases support or challenge the author's positions.

The next chapters cover interviews with magistrates, lawyers, six psychiatrists and probation officers. That relating to psychiatry is little more than a reiteration of well founded and well known complaints: that psychiatrists may use the medical model inappropriately, that they may claim expertise they do not possess, and that they may fail (negligently?) to deliver on promised treatment when there is a condition of probation. If the book had examined the role of credentialling, especially for psychiatrists, some of whom claim forensic expertise they do not possess, then it may have addressed its aim. Regretfully, the author's conversations with six psychiatrists are not interpreted in the light of the need for training or standards (only two are forensic psychiatrists) but rather as evidence that these six misunderstand women offenders.

The book fails as a clear case study. More importantly, its methodological shortcomings mean that it fails in its expressed aim.

PETER L. G. JENKINS, *Lecturer in Psychological Medicine, University of Wales College of Medicine, Cardiff*

Modern Perspectives in the Psychiatry of the Affective Disorders (Modern Perspectives in Psychiatry, Number 13). Edited by JOHN G. HOWELLS. USA: Brunner/Mazel Publishers. 1989. 440 pp. $60.00.

Of all the topics chosen for the *Modern Perspectives in Psychiatry* series, affective disorder should provide the most rapidly ripening harvest. Each of the 38 contributors, chosen as experts in their field, offer much fact and theory, with a wide variation in depth of approach, theoretical framework and perspective. There is also a variety of tone which includes political worldliness, personal research and practical clinical wisdom.

The three section headings are "General topics", "Clinical syndromes" and "Management". The opening chapter on epidemiology of depression is fully comprehensive and concentrates on recent possible factors. Neurochemistry is covered in an excellent essay, giving realistic conclusions, but making a distinction between circadian rhythm disturbance and dysregulation as aetiological pathways. Important new areas are explored including the link between immunity and depression, and the role of thyroid function in the illness. Social psychiatry is reviewed broadly, concluding with an ideal for future psychiatric potential. The pronouncement of DSM–III–R seemed to cause as much concern as the status quo before this. No less than three chapters cover nosology, diagnosis and DSM–III in relation to affective states.

The clinical section focuses on the 'difficult' aspects such as schizoaffective states, atypical depression, postnatal depression and suicide in children and in adults, while mania, involutional melancholia and bipolar affective disorder bring one back to more familiar clinical experiences. This section closes with an approach to prevention. Primary, secondary and tertiary levels are clearly described, but the wide range of possible interventions based on aetiology is not expanded here.

The management section includes firm practical advice on drug treatment and, separately, on psychotherapy including its limitations and the importance of assessing physical factors as well. A review of the association between tardive dyskinesia and affective disorder deals with important risk factors which should be widely noted. A chapter on the use of electroconvulsive therapy (ECT) is excellently summarised, the techniques being particularly well described. Its practical value is enhanced by the inclusion of drug interactions often overlooked. The concluding chapter is a welcome 'hands on' guide to enhancing the therapeutic effect of antidepressants with the addition of lithium.

This work includes most of psychiatry's many disciplines. Consequently, perspective and focus must be adjusted periodically, although movement through the chapters remains smooth. This gives the book a distinct advantage – even when read straight through, it never loses its capacity to appeal, to stimulate or to 'update'.

JOHN POLLITT, *Honorary Consulting Physician in Psychological Medicine, St Thomas' Hospital*

Familial Alzheimer's Disease – Molecular Genetics and Clinical Perspectives. Edited by GARY D. MILNER, RALPH RICHTER, JOHN P. BLASS, JIMMIE L. VALENTINE and LINDA A. WINTERS-MINER. New York & Basel: Marcel Dekker Inc. 1989. 444 pp. $150.00.

The origin of this book is not made clear in the preface, although there is a suggestion that it is part of the proceedings of the first international symposium on familial Alzheimer's disease held in Tulsa in 1987. If the book is the product of this meeting, it is better than the usual conference proceedings. The papers are of a high standard and are sufficiently long to provide some background, explain advances in technique, and discuss methodological problems in a particularly clear way. Most of the papers review the work of others as well as giving original data, and they include an extensive bibliography.

The book is divided into four parts: background of familial Alzheimer's disease and clinical perspective; molecular genetics of Alzheimer's disease; promising new avenues of research; and overview and directions for the future.

The first two parts of the book, which deal with genetics and molecular pathology, are particularly good and are concerned with familial Alzheimer's disease alone. The chapter on genetic heterogeneity is especially good. In part three there is less emphasis on familial Alzheimer's disease, and a number of chapters refer instead to senile dementia of the non-familial type (e.g. the chapter on methionine-enkephalin). Part four contains a fascinating account of the complex organisation set up by a Canadian multidisciplinary team to establish a register of Alzheimer's disease in a defined area. It details the steps taken to identify possible cases as well as documenting their investigation and follow-up up to and including post-mortem.

The book concludes with an appendix containing a research bibliography of familial Alzheimer's disease. This shows the expansion in publications from 1907 to 1988 and belies Khachaturian's statement, in his chapter on "New challenges in Alzheimer's disease and dementia research", that the systematic study of Alzheimer's disease "began only in the last decade".

The book is well produced and the figures are all of a high quality. Because of its price it is most likely to be purchased by libraries, where it will provide a useful account for all those interested in Alzheimer's disease and its rarer familial form.

C. Q. MOUNTJOY, *Consultant Psychiatrist, St Andrew's Hospital, Northampton*

Royal Rebel. A Psychological Portrait of Crown Prince Rudolf of Austria-Hungary. By JOHN T. SALVENDY. New York & London: University Press of America. 1988. 255 pp. £25.85 (hb), £13.75 (pb).

"Tis strange – but true; for truth is always strange: stranger than fiction." This quotation from Byron's *Don Juan* is singularly apropos in the context of the appalling tragedy that was enacted in the hunting lodge at Mayerling on 30 January 1889. In the morning of that day the bodies of the 17-year-old countess Mary Vetsara and the 30-year-old Crown Prince Rudolf of Austria-Hungary were found, the former murdered, the latter dead by his own hand – or so it seemed.

In the ensuing century this evergreen scenario has been endlessly exploited by the media, yielding many different interpretations of the events. By far the most popular is the romantic version in which the prince and his young mistress are portrayed as star-crossed lovers, doomed to separation this side of eternity by impassable barriers. Their union could only be achieved in the hereafter: hence the suicide pact.

According to Salvendy in his exciting and well researched book, this version has no substance in fact. He makes an excellent case, based largely on psychiatric considerations, overlaid with more than a modicum of psychoanalytic theory, that Rudolf was doom-laden from the moment of his conception. His heredity was tainted, and the environment in which he was brought up only served to aggravate the flaws. He was thwarted at every turn by a despotic, insensitive father and an uncaring and, for the most part, absent mother. There were no surrogates, emotionally speaking. His marriage to a dull, unsophisticated young girl was predictably disastrous. He resorted to extra-marital affairs, in the pursuit of one of which he contracted gonorrhea of a most virulent variety which rendered him and his wife, whom he in turn infected, sterile.

He became an irrelevance. He had not sired, and now could never sire, a male heir. There was no hope of him succeeding to the imperial throne in the foreseeable future. There was no outlet for his literary or political propensities. He was in constant discomfort or frank pain from the sequelae of his chronic venereal infection. He lived his life in the abyss. That he became depressed is not surprising and in his despair it is understandable that he took to drink, morphine – and women.

Salvendy maintains that Rudolf had planned his suicide months before the event. He was not emotionally involved with Mary Vetsara, although she, an *ingenue*, was flattered by a relationship with someone as exalted as a Crown Prince. Rudolf needed her co-operation, however, in the suicide pact in order to strengthen his own resolve. He shot her, and some hours later, after writing a series of farewell letters, destroyed himself. Or did he?

One theory, to which Salvendy apparently gives no credence, is that the killings were the result of a deep-laid plot set in motion by reactionary noblemen in order to eliminate a rebellious, liberally minded, nonconformist heir-apparent.

How much then of this spine-chilling saga is truth; how much fiction? In the final analysis, you pays your money and you makes your choice.

HENRY R. ROLLIN, *Emeritus Consultant, Horton Hospital, Epsom*

Not Always on the Level. By E. J. MORON CAMPBELL. London: British Medical Journal. 1988. 246 pp. £14.95.

The *Memoir Club* is a series of handsomely produced volumes by medical authors, written at a sufficiently non-technical level to interest the general reader. This particular one is by a professor of medicine, who suffers from bipolar affective disorder – the bad fairy at his birth which he describes with such graphic imagery that few psychiatrists will fail to learn from the account. Professor Campbell says the condition had dominated his life for the previous 18 years, although he coped pretty well with a weekly cycle of mood swings during the first eight of them. The next seven years, though, were a 'sort of existence' in which he was mostly depressed, between short bursts of hypomania which caused his admission on three occasions. A couple of nights with less than two hours sleep would indicate that trouble was coming Looking back, he realises that he already showed cyclothymia by the age of 20 and that this should have been clearly apparent by the time he was 40; however, until middle-age, his only disability was the occasional spell of depression or insomnia.

The author is right that the everyday life of a country doctor in the 1930s (which his father was) and that of a medical student, houseman and clinical scientist in the 1940s (which he was himself) ought to be described before their memory vanishes, as things have since changed so utterly. Today's student would be unlikely to tolerate a freezing bedroom, filthy kitchen, sheets changed about once a term and – not surprisingly – persistent bed-bugs. Nor would today's houseman accept from his boss that "I expect you to remain in the hospital at all times. You have no off-duty". This rather unedifying start to clinical work was followed by research in physiology, at a time, the author says, when respiratory failure was scarcely recognised. He became an expert on breathlessness, leading to the Foundation Chair of Medicine at McMaster and, as is not uncommon in the vulnerable, achievement of a major ambition was then followed by severe depression, and in turn by mania.

Piling Pelion on Ossa, another manic episode was the cause of a severe road accident, in which his wife suffered particularly badly. This time in the relative's role, Professor Campbell makes trenchant criticisms of the culture of intensive care units, finding that whatever their technological sophistication, no one doctor would assume overall responsibility for the patient and no one told him what was happening to her. Mania stepped in again, when he was invited to return to London but became psychotic at the critical time.

Latter parts of the book were written while he was actually experiencing manic episodes and offer some insights into the upside-down logic that provokes their characteristic behaviour. Reading such an account, the armchair clinician should resist the temptation to believe that he could have done better than his colleagues on the job, yet when the author says of his condition that "drugs have not worked", it is hard to escape a feeling that perhaps they did not get a proper chance – difficult as he must have been as a patient.

This very frank story brings home the havoc that can still be wreaked on an individual and his family by severe affective illness. At the same time, the author is able to describe two cases in which his own determined efforts and clinical nous saved the lives of patients who had been given up by luminaries of the profession. That is not a bad record for any medical career.

HUGH FREEMAN, *Editor, British Journal of Psychiatry*

The Medical Evaluation of Psychiatric Patients. By RANDOLPH SCHIFFER, ROBERT F. KLINE and ROGER C. SIDER. New York & London: Plenum. 1989. 247 pp. $35.00.

Psychiatrists in most branches of the profession should find this book very useful. Rather than evaluate the psychopathology of organic disorders directly, it deals in a very practical way with the differential diagnosis and investigation of potential organic causes of common psychiatric conditions.

Doubts over the diagnosis of atypical cases of, for example, anxiety, depression or mania, are often not fully relieved by available textbooks. How many of us could name 46 causes of secondary mania? What is the detailed differential diagnosis of organic causes of paranoid psychoses or of physical fatigue? How should hypoparathyroidism be detected? It is for extensive answers to those kinds of questions, applied in a psychiatric context, that this book is valuable.

The authors impart a strong sense of practicality and experience to the text. Discussion of the causes of a wide variety of psychiatric symptoms, the description of numerous procedures and investigations, the provision of clinical examples, evident good judgement and plentiful references make it enjoyable to read and create a sense of diagnostic sufficiency.

The only section which may seem a little odd to clinicians trained to eclectic psychiatric practice with an organic flavour is the first chapter where the case for examining and investigating patients is pleaded with an almost desperate air. Breaking psychoanalytic taboos by palpating the patient's body or taking blood is still evidently difficult in some quarters despite the implications of negligence.

CHRISTOPHER WHITE, *Senior Lecturer, University Department of Psychiatry, Ninewells Hospital, Dundee*

The Future of Mental Health Services Research. Edited by CARL A. TAUBE, DAVID MECHANIC and ANN A. HOHMAN. Washington: US Government Printing Office. 1989. 324 pp.

This volume consists of papers presented at a conference on the future of mental health services research held in Florida in February 1987. The main themes were the structure and organisation of systems providing mental health services; costs, financing, reimbursement and regulation; determinants and patterns of service use; outcome of service use; and the definition, classification and measurement of mental illness and services.

Two ideas immediately came to mind as I began to read. First, how refreshing it is to hear research managers in the United States being so up-beat about the possibilities for mental health research and funding. Secondly, the telling words of a former colleague: health service research is intellectual death.

In the event, the Americans discussed a range of issues important enough to stimulate and encourage even European observers. Perhaps of particular current interest to British psychiatrists are articles on the pursuit of quality services and the effectiveness of services for people with severe mental illness. Professor Morrissey with the final word, identified four specific topics as future research priorities: conceptualisation and measurement of severity of illness; content of services; measurement and analysis of service packages; and system of care as a unit of analysis.

We have a long way to go to match the approach taken by the NIMH in these matters. Regrettably, this valuable resource may be difficult to get hold of, but it is "for sale by the Superintendent of Documents, US Government Printing Office, Washington DC 20402".

GREG WILKINSON, *Director of the Academic Sub-Department of Psychological Medicine in North Wales, North Wales Hospital, Clwyd.*

Contents of
THE AMERICAN JOURNAL OF PSYCHIATRY

Volume 147, Number 7 July 1990

Special Articles

823 Hypnotizability and dissociation. *Fred H. Frankel*

830 A review of the depressive personality. *Katharine A. Phillips, John G Gunderson, Robert M A. Hirschfeld and Lauren E. Smith*

838 Anxiety and cerebral blood flow *Roy J Mathew and William H. Wilson*

Regular Articles

850 Symptom criteria and family history in major depression. *Daniel N. Klein*

855 Alzheimer's disease and depression· neuropsychological impairment and progression of the illness *Oscar L. Lopez, Francois Boller, James T. Becker, Mark Miller and Charles F. Reynolds III*

861 Symptoms of major depression among Mexican-Americans and Non-Hispanic whites. *Jacqueline M Golding, Marvin Karno and Carolyn M Rutter*

867 "Anger attacks": possible variants of panic and major depressive disorders. *Maurizio Fava, Keith Anderson and Jerrold F. Rosenbaum*

871 Maintenance treatment and 6-month outcome for bulimic patients who respond to initial treatment. *Richard L. Pyle, James E. Mitchell, Elke D. Eckert, Dorothy Hatsukami, Claire Pomeroy and Robert Zimmerman*

876 A double-blind, placebo-controlled trial of fluoxetine plus behavior modification in the treatment of obese binge-eaters and non-binge-eaters. *Marsha D. Marcus, Rena R Wing, Linda Ewing, Edward Kern, Michael McDermott and William Gooding*

882 CSF oxytocin in anorexia nervosa and bulimia nervosa: clinical and pathophysiologic considerations. *Mark A. Demitrack, Michael D. Lesem, Sam J. Listwak, Harry A. Brandt, David C. Jimerson and Philip W. Gold*

887 Dissociative symptoms in relation to childhood physical and sexual abuse. *James A. Chu and Diana L. Dill*

893 Ventricular abnormalities in affective disorder: clinical and demographic correlates. *Nancy C. Andreasen, Victor Swayze II, Michael Flaum, Randall Alliger and Gregg Cohen*

901 Allergic to life psychological factors in environmental illness. *Gregory E. Simon, Wayne J. Katon and Patricia J Sparks*

907 Effect of menstrual cycle phase and oral contraceptive use on serum lithium levels after a loading dose of lithium in normal women *Susan Chamberlain, Philip M. Hahn, Peter Casson and Robert L. Reid*

910 Psychiatrists' beliefs about gender-appropriate behavior. *Marcia J Kaplan, Carolyn Winget and Noel Free*

913 The prevalence of posttraumatic stress disorder and its clinical significance among Southeast Asian refugees. *J. David Kinzie, James K. Boehnlein, Paul K Leung, Laurie J Moore, Crystal Riley and Debra Smith*

918 Identifying criteria for narcissistic personality disorder. *Elsa Ronningstam and John Gunderson*

923 Sertraline in obsessive-compulsive disorder: a double-blind comparison with placebo *Michael A. Jenike, Lee Baer, Paul Summergrad, William E. Minichiello, Amy Holland and Ralph Seymour*

929 The hypothalamic-pituitary-thyroid axis in social phobia *Manuel E. Tancer, Murray B. Stein, Cheryl S. Gelernter and Thomas W. Uhde*

933 Autonomic orienting responses in 15-year-old male subjects and criminal behavior at age 24. *Adrian Raine, Peter H. Venables and Mark Williams*

Contents of The Journal of Nervous and Mental Disease

July 1990 **Volume 178, Number 7**

Diagnostic and research applications of electroencephalographic sleep studies in depression. Conceptual and methodological issues. *Daniel J. Buysse and David J. Kupfer* 405

Supportive housing for the chronically mentally ill. Matching clients with community environments. *Jill M. Goldstein, Joseph F. Dziobek, Robin Clark and Ellen L. Bassuk* 415

Physical recklessness in adolescence. Trait or byproduct of depressive/suicidal states? *David C. Clark, Lorraine Sommerfeldt, Marvin Schwarz, Donald Hedeker and Lucie Watel* 423

Racial differences in prevalence of phobic disorders. *Diane R. Brown, William W. Eaton and Linda Sussman* 434

Psychopathology among cocaine abusers entering treatment. *Paula H. Kleinman, Amy B. Miller, Robert B. Millman, George E. Woody, Thomas Todd, Jack Kemp and Douglas S. Lipton* 442

Characteristics of patients with multiple personality and dissociative disorders on psychological testing. *Judith G. Armstrong and Richard J. Loewenstein* 448

Electroconvulsive therapy emergence agitation and succinylcholine dose. *Conrad M. Swartz* 455

Brief Reports

Resolution of psychotic depression after right temporoparietal infarction. 458
Delusional depression responding to clomipramine in Binswanger's disease. 459
Stress, social support, coping resources, and immune status in elderly women 460

Book Reviews. Gay, P. *Freud: A Life for Our Time;* Lidz, T. and Lidz, R. W. *Oedipus in the Stone Age: A Psychoanalytic Study of Masculinization in Papua New Guinea;* Caper, R. *Immaterial Facts: Freud's Discovery of Psychic Reality and Klein's Development of His Work;* Sjöbäck, H. *The Freudian Learning Hypotheses: Psychoanalytic Ideas about the Scope and Nature of Early Environmental Influence on Character Development and Pathogenesis;* Noblit, G. W. and Hare, R. D. *Meta-Ethnography: Synthesizing Qualitative Studies;* Basch, M. F. *Understanding Psychotherapy: The Science behind the Art;* Ornstein, R. and Ehrlich, P. *New World New Mind: Moving toward Conscious Evolution;* Kubicki, S., Henkes, H., Bienzle, U. and Phle, H. D. *HIV and the Nervous System: Proceedings of the Symposium on Neurological Aspects in AIDS;* Dunner, D. L., Gershon, E. S. and Barrett, J. E. (eds). *Relatives at Risk for Mental Disorder;* Friedman, R. C. *Male Homosexuality: A Contemporary Psychoanalytic Perspective* 462

Index to Advertisers

Pharmaceuticals
Duphar Laboratories .... *facing* p 239
Janssen Pharmaceuticals Ltd. .... p viii
Janssen Pharmaceuticals Ltd. .... *facing* p 261
Janssen Pharmaceuticals Ltd. .... *facing* p 264
Lederle Laboratories Ltd. .... *facing* p 238
Lederle Laboratories Ltd. .... *facing* p 264
Lundbeck Limited .... inside front cover
Lundbeck Limited .... outside back cover
E. Merck Ltd. .... *facing* p 260
Rhone-Poulenc .... *facing* p 238

Appointments
American University of Beirut .... p vi
Dalhousie University .... p ii
Medicine Hat Regional Hospital .... pp iv & vi

Booksellers & Publishers
American Psychiatric Press .... p vii
Nordic Journal of Psychiatry .... p v

Clinics, Hospitals & Nursing Homes
St. Andrew's Hospital .... *facing* p 265

Miscellaneous
NHS Training Authority .... p ii
ROTA .... p vi
St. George's Hospital Medical School .... p iv

Printed in Great Britain by Henry Ling Ltd., at the Dorset Press, Dorchester, Dorset

STORMS BLOW
FIND THE EYE
WINDS CHANGE
... REFLECT (Anon)

CLOPIXOL®
NEW ACUPHASE
zuclopenthixol acetate ▼

A harmonious new way to treat acute psychosis

Lundbeck

FACING THE FUTURE TOGETHER

Further information is available on request from

30. 4. 91

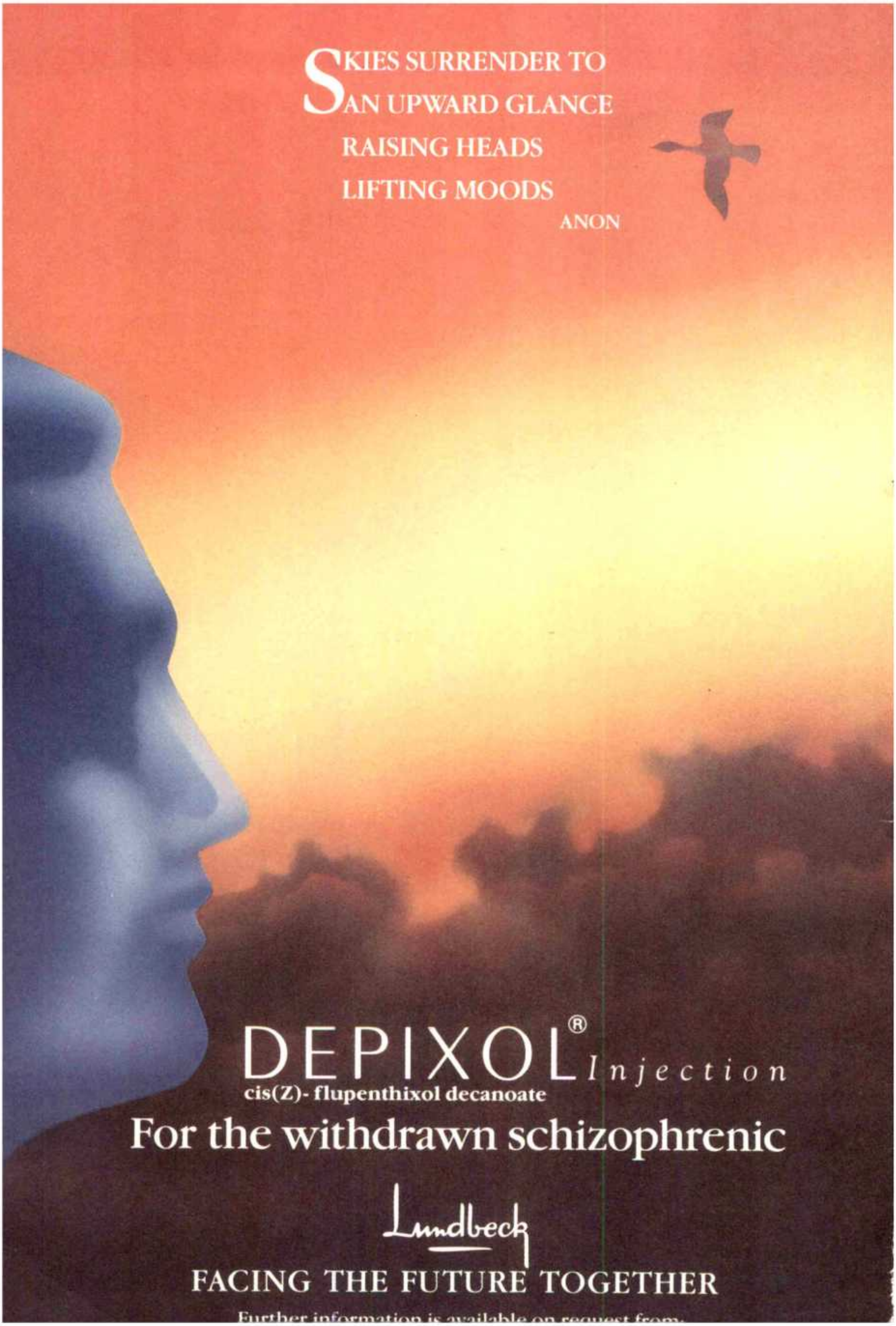
THE BRITISH JOURNAL OF PSYCHIATRY

September 1990 Vol 157

CALCUTTA UNIVERSITY CENTRAL LIBRARY

Published by
The Royal College of Psychiatrists

ISSN 0007-1250


SKIES SURRENDER TO
SAN UPWARD GLANCE
RAISING HEADS
LIFTING MOODS
ANON
DEPIXOL® Injection
cis(Z)- flupenthixol decanoate
For the withdrawn schizophrenic
Lundbeck
FACING THE FUTURE TOGETHER
Further information is available on request from:

THE BRITISH JOURNAL OF PSYCHIATRY

September 1990 Volume 157

Annotation

Morbid jealousy: a review and cognitive-behavioural formulation. *N. Tarrier, R. Beckett, S. Harwood and N. Bishay* 319

Comment

Treatment of Alzheimer's disease. Molecular pathology versus neurotransmitter-based therapy. *D. M. Bowen* 327

Papers

Stress and puerperal psychosis. *I. F. Brockington, C. Martin, G. W. Brown, D. Goldberg and F. Margison* 331
Is psychiatric training still improving? *P. Brook* 335
Psychiatric diagnoses as predictors of suicide. A comparison of diagnoses at conscription and in psychiatric care in a cohort of 50465 young men. *P. Allebeck and C. Allgulander* 339
Schizophrenia and crime A longitudinal follow-up of 644 schizophrenics in Stockholm. *P. Lindqvist and P. Allebeck* 345
Langfeldt's schizophreniform psychoses fifty years later. *A. L. M. Bergem, A. A Dahl, C. Guldberg and H Hansen* 351
Impaired ability of schizophrenics, relative to manics or depressives, to appreciate social knowledge about their culture. *J Cutting and D. Murphy* 355
Regional cerebral blood flow in patients with affective disorders. *V. Delvenne, F. Delecluse, Ph. P. Hubain, A. Schoutens, V. De Maertelaer and J. Mendlewicz* 359
Dichotic perception and memory following electroconvulsive treatment for depression. *K. M. Williams, W. G. Iacono, R. A. Remick and P. Greenwood* 366
Towards a cognitive-behavioural intervention for adult grief reactions. *D. J. Kavanagh* 373
The value of the Claybury selection battery in predicting benefit from group psychotherapy. *M. J. Pearson and A. J. Girling* 384
Two-year follow-up of the Sheffield psychotherapy project. *D. A. Shapiro and J. Firth-Cozens* 389
A systematic inquiry into recollections of childhood experience and their relationship to adult defence mechanisms *E. Tauschke, H. Merskey and E. Helmes* 392
Incest, incestuous fantasy and indecency. A clinical catchment area study of normal weight bulimic women. *J. H Lacey* 399
Hypochondriasis and somatosensory amplification. *A. J. Barsky and G. Wyshak* 404
Are British psychiatrists racist? *G. Lewis, C. Croft-Jeffreys and A David* 410
A comparative study of psychiatric services in Japan and England. *Y. Mino, R. Kodera and P. Bebbington* 416

The Current Literature

Mass sociogenic illness by proxy. parentally reported epidemic in an elementary school. *S. Wessely and C. J. Wardle* 421

Brief Reports

The depressed elderly living in the community. A follow up study. *O. Ben-Arie, M. Welman and A. F. Teggin* 425
Post-partum psychosis in the Assir region of Saudi Arabia. *I. H. Shoeb and G. A. Hassan* 427
Panic attacks in chronic schizophrenia. *N. Argyle* 430
Schizophrenia and Marfan syndrome. *P. Sirota, M Frydman and L. Sirota* 433
Carbamazepine and forme fruste neuroleptic malignant syndrome. *T Dalkin and A. S. Lee* 437
Klüver-Bucy syndrome and psychiatric illness. *D. J. Clarke and N. S. Brown* 439
Mood disorder, 'pre-ictal' psychosis and temporal lobe damage. *B. A. Johnson and L. B. Campbell* 441
Suicidal behaviour in severely mentally handicapped patients. *R. M. Walters* 444

Correspondence 447

A Hundred Years Ago. Researched by Henry Rollin 460

Reading About... Neuropsychology. *A. Costello* 461

Book Reviews 464

American Journal of Psychiatry, Volume 147, Number 8, August 1990 474

The Journal of Nervous and Mental Disease, Volume 178, Number 8, August 1990 *facing* 474

(N.B. With effect from January 1991, the contents list will be moved to the cover of the *Journal*).

Royal College of Psychiatrists

Gaskell

Recent books from Gaskell

Psychiatry Around the Globe: A Transcultural View (2nd edition). By Julian Leff. The aim of this text is to provide a transcultural perspective on psychiatry It presents five main issues: the form, frequency, treatment, and natural history of mental disorders in different cultures are examined, and the fifth section deals with migration. This second edition has been extensively revised and updated. *ISBN 0 902241 24 9, 226 pp, £10.00.*

Consent and the Incompetent Patient. Edited by Stephen R. Hirsch and John Harris. The conflict between the need to protect vulnerable patients from unwarranted research and treatment on the one hand, and to ensure that they receive the treatment they need and that helpful research is pursued on the other, is discussed by barristers, psychiatrists and representatives of patients. *ISBN 0 902241 22 2, 101 pp, £7.50.*

Aspects of Autism: Biological Research. Edited by Lorna Wing. The evidence for a physical cause for autism is extensively reviewed, and the roles of endogenous opioids, genetics, serotonin, urinary peptide metabolites, intrauterine cytomegalovirus plus antibodies, and magnesium + vitamin B6, folates and fenfluramine are examined. *ISBN 0 902241 25 7, 120pp, £7.50.*

Interviewing the Sexually Abused Child. By David P. H. Jones and Mary G. McQuiston. This guide for those who need to know whether a child has been sexually abused focuses on the evaluative interview, giving guidelines on its structure, discussing the use of language, free play, toys and play materials, and examining the importance of interviewer style. *ISBN 0 902241 23 0, 56pp, £4.50.*

Drug Scenes. A report on drugs and drug dependence by the Royal College of Psychiatrists. This report lays out the facts on the nature and causes of addiction, describes the wide range of problems which can arise from misuse of different substances, debates a series of contentious issues, and puts forward a set of challenging recommendations. *ISBN 0 902241 18 4, 226pp, £5.95.*

Psychiatric Beds and Resources: Factors Influencing Bed Use and Service Planning. By a Working Party of the Royal College of Psychiatrists (Chairman S. R. Hirsch). This report reviews the literature and presents important new data showing a close relationship between social factors and the uptake of psychiatric services. An approach to planning, based on analysis of service provision, utilisation, and potential demand, is demonstrated. *ISBN 0 902241 21 4, 82pp, £7.50.*

Available from your bookseller or, in case of difficulty, send order with payment to the Publications Manager, Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG.

The British Journal of Psychiatry

September 1990, Volume 157

Published monthly. Founded in 1853 as the *Asylum Journal* and known as the *Journal of Mental Science* from 1855 to 1963

Editor: Hugh Freeman

Senior Associate Editor
Alan Kerr

Associate Editors
Dora Black, Sidney Crown, Julian Leff, Sir Martin Roth, Peter Tyrer

Assistant Editors
Mohammed Abou-Saleh, Peter Bruggen, Patricia Casey, Alexander Kellam, Stuart Montgomery, Catherine Oppenheimer, Ian Pullen, Rosalind Ramsay, Henry Rollin, Oliver Russell, George Stein, Digby Tantam, Greg Wilkinson, Stephen Wilson

Publications Manager
Dave Jago

Scientific Editor
Ralph Footring

Bulletin Manager
Elaine Millen

Assistant Scientific Editor
Katy Wimhurst

Editorial Assistants
Judy Ashworth, Ian O'Reilly, Christine Connolly

⊗™ The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences—Permanence of Paper for Printed Library Materials. ANSI Z39. 48–1984.

Printed by Henry Ling Ltd, The Dorset Press,
23 High East Street, Dorchester, Dorset DT1 1HD
(tel. 0305-251 066).

©1990 The Royal College of Psychiatrists

Subscriptions

Non-members of the College should contact the British Journal of Psychiatry Subscription Department, The Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. Annual subscription rates for 1990 (12 issues post free) are as follows:

UK: institutions £105, individuals £86
US: institutions $265, individuals $162
Elsewhere: institutions £132, individuals £106
Single copies of the Journal, £10, $20 (post free)

Payment should be made out to the British Journal of Psychiatry.

Queries from non-members about missing or faulty copies should be addressed within six months to the same address; similar queries from College members should be addressed to the Registration Department, The Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG.

Advertising

Correspondence and copy should be addressed to Peter T. Mell, Advertisement Manager, PTM Publishers Ltd, 282 High Street, Sutton, Surrey SM1 1PQ (tel. 081-642 0162, fax 081-643 2275).

US Mailing Agents: Mercury Airfreight International Ltd. Inc., 2323 Randolph Avenue, Avenel, NJ 07001.

Back issues published before 1988 may be purchased from William Dawson & Sons Ltd, Cannon House, Folkestone, Kent, England (tel. 0303-850-101).

A CANADIAN OPPORTUNITY

MEDICINE HAT REGIONAL HOSPITAL

requires a

PSYCHIATRIST

The Position: To work on a sessional basis as a Psychiatrist on the Adult Psychiatric Services of the Hospital. Will be expected to work closely with the Chief of Psychiatry in developing services in the community.

The Hospital: Medicine Hat Regional Hospital is a district general hospital with a modern 26 bed Psychiatric Unit with a Day Hospital Program. Psychogeriatric and Child Psychiatric Services and provided by other psychiatrists working on sessional basis.

The Location: Medicine Hat is located in the South Eastern corner of Alberta. The Hospital has a catchment area of 79,000. A wide range of recreational facilities are available.

Qualifications. Minimum MRCPsych. Graduates of Medical Schools other than United Kingdom, Australia and New Zealand may require special qualifications.

Visa: Assistance will be provided to the successful candidate towards obtaining the necessary Visa.

Remuneration. Approx. CA$150,000.

Interviews. Preliminary interviews will be held in London, England in September or October.

Apply with CV and names of three references in confidence to:
Dr S. Nandy, Chief of Psychiatry, Medicine Hat Regional Hospital,
666 – Fifth Street S.W., Medicine Hat, Alberta, Canada T1A 4H6
Tel. 403-529-8036: Fax. 403-529-8010.

COURSE

Child Abuse & Neglect
Preparation for Court – Children's Act

A 3-day course commencing 10, 11 & 12 October, 1990 – Postgraduate Centre, Southmead Hospital, Bristol, covering·

Problems Related to Child Abuse, Sexual Abuse, Communicating with Children, Collecting Forensic Evidence, Preparation of Court Reports, Court Proceedings and Pitfalls, After Cleveland.

One half day will be devoted to the Children's Act – This can be booked separatedly

Limited places offered on a first come first served basis.

The Course fee: (For the 3 days) £180 which includes Buffet Lunch/Refreshments
(For the Half Day on Children's Act) £40

For further details and an application form please contact:
Dr Jean Price, Consultant Community Paediatrician, Community Child Health Services, Westgate House, Southmead Hospital, Westbury-on-Trym, Bristol BS10 5NB. Tel:0272 505050. Ext: 3620.

PSYCHIATRISTS
NEW BRUNSWICK
CANADA

The Mental Health Commission of New Brunswick has at present several vacancies for Psychiatrist M R C P Psych (U K) required.

Positions are available on both child and adult psychiatry in community mental health clinics and psychiatric hospitals. Representatives from the Mental Health Commission of New Brunswick will be travelling to the United Kingdom to interview interested applicants within the next few months.

A competitive salary and a full range of employee benefits, including personal development and education opportunites are available.

Assistance in relocating and immigration will also be offered to the successful candidates

If you are interested in further exploring this opportunity, please forward a Curriculum Vitae to

Raymond T. Vaudry
Industrial Development Officer
Office of the Agent General of Nova Scotia
14 Pall Mall
London, England SW1Y 5LU

British Journal of Psychiatry (1990), 157, 319–326

Annotation

Morbid Jealousy: A Review and Cognitive–Behavioural Formulation

NICHOLAS TARRIER, RICHARD BECKETT, SUSAN HARWOOD and NAGY BISHAY

Jealousy is a common emotion which can be considered pathological in some circumstances. The term 'morbid jealousy' has been used to signify this abnormal or extreme condition. However, the literature on morbid jealousy is sparse and the empirical data fragmentary. The aims of this paper are to briefly review the concept, to define the dimensions of the disorder and to apply a cognitive-behavioural framework to it. Such a summary paper is timely, to draw together the different aspects of the problem and to provide a theoretical base from which to generate further research.

Conceptualisation of morbid jealousy

The psychiatric approach

Essentially, morbid jealousy has been viewed as a symptom or sign of other psychiatric conditions or illnesses rather than a nosological entity in itself. It has been classified as occurring in association with three types of conditions (Shepherd, 1961): toxic or organic cerebral disorders (including alcoholism, addiction to substances other than alcohol, psychiatric disorders in the senium, and other cerebral disorders); functional psychoses (including schizophrenia and paranoid states, and affective psychoses); and neurotic and personality disorders.

The association between morbid jealousy and alcoholism has a long history (see Shepherd, 1961; Madder, 1979, Shrestha *et al*, 1985). However, it is generally concluded that while alcohol abuse frequently precipitates jealousy and violence, it is rarely the primary cause (Landfeldt, 1961; Shepherd, 1961). Shepherd (1961) also cited morbid jealousy as occurring in amphetamine and cocaine abuse, and occasionally it has been associated with various disorders, usually neurological, of old age.

Delusional jealousy is one of the DSM–III criteria for paranoid disorder (American Psychiatric Association, 1980) and, although many diagnostic systems such as the DSM–III make a separate classification of paranoid psychosis, patients experiencing paranoid delusions would be frequently included within the category of schizophrenia. Delusions concerning the infidelity of the partner are not uncommon in patients suffering from schizophrenia (Abely & Feuillet, 1941).

The fact psychiatric medicine has tended to concentrate on morbid jealousy as a symptom of the psychoses is reflected in the treatment approaches (Mooney, 1965; Pollock, 1982; Munro, 1984).

The behavioural approach

There appears to be little in the behavioural literature concerning morbid jealousy. Cobb & Marks (1979) compare morbid jealousy to obsessional–compulsive neurosis, in that some patients with this disorder present with jealous thoughts as ruminations and checking for evidence of infidelity as compulsive rituals.

The sociobiological approach

Daly *et al* (1982) proposed that in species which have evolved internal fertilisation, the males cannot confidently identify their offspring. Because of this, species which have developed paternal investment will defend against expending that investment on unrelated young. Hence, male sexual jealousy is part of a behavioural-motivational complex, also including the use or threat of violence which has evolved to achieve sexual exclusivity and to control the sexual function in order to defend paternity confidence. Here Daly and his colleagues are discussing male jealousy in general. However, they quote the sex distribution of morbid jealousy as evidence that sexual jealousy is a 'ubiquitous aspect of male psychology'.

Problems of definition

The study of jealousy has the problem of attempting a scientific account of a term derived from everyday language. This results in a conceptual 'fuzziness'. This is especially true of jealousy which is morbid or pathological, about which there appears to be an implicit assumption by many authors that what is reasonable and what is unreasonable jealousy will be tacitly understood. Defining parameters of morbid jealousy have not been clearly stated. Like the elephant or pornography, morbid jealousy is easier to recognise than define.

Cobb's (1979) classification is useful since it divides normal from morbid jealousy and then subdivides the latter into neurotic (non-psychotic) and psychotic, giving three categories in total. 'Psychotic' pertains to jealous thoughts being of a delusional nature and the distinction between it and neurotic jealousy has implications for treatment.

Some authors (e.g. Shrestha *et al*, 1985; Shepherd, 1961) use the term to include excessive responses to real infidelity as well as jealousy in response to imagined infidelity. However, this confounds jealousy in terms of suspicion where infidelity is imagined, and anger where it is real.

A definition of morbid jealousy must include a core concept of a belief or suspicion of the partner's unfaithfulness, which has consequential thoughts, feelings or behaviour. However, to enable a distinction to be made between normal and morbid jealousy, the term should be restricted to describe an unfounded preoccupation with the partner's infidelity. Thus, the characteristic of morbid jealousy is that the thoughts are irrational, unsubstantiated and occur out of context. That is, the suspicions concerning the partner's fidelity need not be precipitated or maintained by reliable evidence. The person experiencing these irrational thoughts is reacting to the thoughts themselves and not to the evidence (or lack of it) that supports them. Reactions to jealous thoughts and the thoughts themselves can be generalised across situations, time and 'rivals'. Morbid jealousy will disrupt the functioning of the person who experiences it, and frequently affect their partner and hence the relationship. The jealous thoughts and any associated feelings or behaviour of either sufferer or partner will be perceived as problematic, that is exceeding the levels of possessiveness regarded as the norm for that society or culture.

Conversely, normal jealousy would be expected to be: reality based; specific to partner, event or 'rival'; transient, only persisting as long as the partner's behaviour or infidelity continued; and the partner's behaviour, not the jealousy itself, would be perceived as problematic. Morbid jealousy is therefore a description not about the extent of the jealous response but about the rationality and objectivity which precipitates that response.

The nature of morbid jealousy

Morbid jealousy is a multi-dimensional emotional complex relating to the unfounded suspicion of sexual and emotional rivals and a fear of losing the partner and relationship, manifested as responses in the cognitive, affective and behavioural domains. In this section we will describe how the morbidly jealous individual probably differs from normal in these domains. This should form the basis of a clinical assessment.

In patients not suffering from a psychotic illness this complex emotional response-set is presumed to be learned. In psychotic patients the situation is less clear; the delusions are presumed to be part of the psychotic illness, although this does not exclude a learnt component.

We have adopted the approach that the cognitive events are the core problem and that behavioural and affective sequelae are the primary consequences, the effect on the patient's life, relationship and environment being the secondary consequences. Identifying cognitive events without clearly delineated behaviour is problematic, but a similar approach has been successful with cognitive models of depression and stimulated considerable research and treatment innovation.

Preoccupation

Intrusive thoughts and suspicions about the partner's fidelity are clearly central to the disorder. These would also include thoughts and images about where the partner was, what they had been doing in the past and even what they would prefer to be doing. The frequency, intensity, duration and situation specificity of the thoughts and the ability to dismiss them appear to vary from individual to individual.

These thoughts have similarities with obsessional ruminations (Cobb & Marks, 1979), and automatic thoughts which occur in depression (Beck *et al*, 1979) in that they appear to be generated spontaneously and frequently go unchallenged.

Confirmatory behaviour

Overt behaviours of seeking to confirm these suspicious preoccupations are common and are evident to others, especially the partner. Constant interrogation of the partner about their whereabouts, actions or even thoughts and intentions, accompanied by accusations of infidelity and followed by arguments, are usually present (Doherty & Ellis, 1976). Sufferers will often embark on ritualistic-like behaviour to check on their partner. Telephone calls to work, surprise visits, surreptitious following of the partner, searching of the partner's clothes and possessions, reading of diaries and interception of mail, examination of underclothes and bed linen for evidence of coitus are all common confirmatory behaviours (Shepherd, 1961; Mooney, 1965, Seeman, 1979). Some patients have been described by Shepherd (1961) to "make strenuous efforts to corroborate their suspicions", such as hiring detectives to follow their partners, hiding microphones to detect secret liaisons, making excessive sexual demands to prove their partner is satiated or going to extreme lengths to extract a 'confession' such as demanding their partner submit to a lie-detection test or hypnosis (Shepherd, 1961; Mooney, 1965).

Avoidance

Behavioural avoidance of jealousy-provoking situations frequently appears in the accounts of patients and their partners. Female patients report avoiding magazines or newspapers which contain pictures of young and attractive women. Similarly, television programmes, films and even mention of other women or women's names are also avoided. Both patients and their partners report avoidance of members of the patient's sex, or situations in which these imagined rivals may be encountered. Hafner (1979) noted the occurrence of morbid jealousy in the husbands of agoraphobic women. Strong avoidance would theoretically maintain jealousy by preventing the habituation of jealousy-associated arousal.

Personal distress

Although the literature contains little systematically collected data, anecdotal accounts abound. Patients have frequently described feeling depressed, anxious, irritable, agitated, phobic and sometimes suicidal (Shepherd, 1961; Mooney, 1965; Seeman, 1979, Cobb, 1979; Teisman, 1979, Im *et al*, 1983; Bishay *et al* 1989). Mooney (1965) described non-psychotic jealousy as more likely to be associated with tiredness, depression and suicidal tendencies than psychotic jealousy.

Disruption to functioning

Constant jealous ruminations and their primary consequences will disrupt the patient's functioning by competing with and inhibiting other on-going and goal-directed behaviour. Their effects on the patient's and/or partner's behaviour and the disruption to their emotional and sexual relationship are well documented (e.g. Shepherd, 1961; Seeman, 1979; Teisman, 1979; Coen, 1987; Doherty & Ellis, 1976; Im *et al*, 1983).

Violence

Arguments and accusations can frequently result in verbal and physical violence and sometimes murder (Doherty & Ellis, 1976). The behavioural expression of jealousy has attracted the attention of forensic workers in two related fields, spouse abuse and homicide.

There is a consensus among authors that the actual occurrence of violence is much higher than that known or treated (Stahly, 1978). Women, who represent the vast majority of victims, rarely present at clinics with explicit complaints of spouse abuse. It has been found that as the intensity and frequency of violent episodes increase, inappropriate adaptations to the situation occur. Partners of aggressively jealous men develop a range of symptoms including feelings of helplessness, isolation, extreme passivity, anxiety, depression, and abuse of prescription drugs and alcohol. It is often the latter complaints and 'accidental' physical injuries that bring women to clinics (Gayford, 1975; Hilberman & Munson, 1978). Jealousy has emerged in empirical studies as a major factor in the cause of conflict among couples and the precipitation of spouse abuse and violence (Hilberman & Munson, 1978; Rounsaville, 1978; Daly *et al*, 1982).

Jealous assaults typically occur within a general climate of verbal demands and threats, confrontation and arguments, and restriction placed upon the spouses' social and work activities. This produces 'anticipatory terror' (Hilberman & Munson, 1978), helps shape both partners' behaviour, and accounts for the development of anxiety and depressive disorders which are commonly reported in the literature on spouse abuse.

As with spouse abuse, jealousy has also emerged as a significant contributary factor in the cause of homicides (e.g. West, 1968; Wilt, 1974; Gillies, 1976). The above studies rely upon the analysis of police records whose primary objective is to identify the events immediately preceding the homicide. However, Chimbos (1978) and Guttmacher (1955) suggest that police files seriously underestimate the prevalence of sexual jealousy as a homicide motive.

Aggression to third parties

This refers to accusations, argument and violence towards a third party, who, in the absence of any objective evidence, is perceived as being at worst the partner's lover, and at best a potential rival. There appears to be little systematically collected data on this dimension, but partners of morbidly jealous individuals frequently cite its occurrence.

Delusions

The psychiatric literature has given greatest attention to delusions of jealousy, and some authors equate morbid jealousy with a delusional state (e.g. Enoch & Trethowan, 1979). It is possible that the non-deluded patient rarely comes into contact with the psychiatric services, which could explain the dearth of interest and data.

Insecurity, lack of confidence and poor self-esteem

Low self-esteem, feelings of insecurity and inferiority, poor self-image and poor self-assertion are characteristics of the morbid jealousy patient that constantly appear in the literature (Shepherd, 1961; Seeman, 1979; Cobb, 1979). During clinical interview, patients will frequently describe poor opinions of their worth, feelings of being unattractive, and low confidence within the context of their emotional and sexual relationships, even if the opposite feelings are true of other areas of their lives.

A cognitive-behavioural formulation of non-psychotic morbid jealousy

The previous review of morbid jealousy suggests that any formulation of the phenomena must account for a variety of features which present in the non-psychotic, morbidly jealous individual. In addition to the affective component (e.g. jealousy), a model should incorporate an explanation of why jealous behaviour continues in the absence of objective evidence. Moreover, it should account for why the behavioural expression of jealousy varies both across individuals and within the same individual across time, as well as why, in some circumstances, it is associated with guilt and depression and in others with anger and aggression.

It is proposed that the central feature of morbid jealousy is the individual's propensity to make systematic distortions and errors in the perception and interpretation of events and information. As such, events which have the potential for misinterpretation will provoke jealousy in subjects whose cognitive expectations and appraisals produce a bias towards particular and erroneous interpretations.

The most influential model of psychopathology to focus on cognitive processes has been developed by Beck and his co-workers, especially in relation to depression (Beck, 1967; 1976; Beck *et al*, 1979). In this model distorted cognitions or automatic thoughts are produced when an individual's unrealistic schemata of underlying beliefs and assumptions are activated by a salient event.

Automatic thoughts are part of the 'stream of consciousness' that tend to be in an individual's awareness. They are fairly labile, elicited by external or internal stimuli and their content is generally associated with a particular affect or behaviour (Beck, 1983). Thus, in morbid jealousy patients these thoughts would probably be associated with the loss or fear of the loss of the partner to a rival or loss of the relationship through the partner's infidelity.

Schema are more stable (although still state-dependent), underlying beliefs or assumptions of a general nature which concern the world and the patient's relation to it. These assumptions are possibly formed from past experiences and serve to direct the patient's attention to current events. In jealous patients the schema probably relate to general and erroneous assumptions about relationships and human sexual and emotional behaviour. In a similar manner to the depressed patient, the jealous patient makes errors in logic which involve systematic distortions in the interpretation of information. For example, a woman who holds an assumption that every man will have an affair if given the opportunity, will have suspicious thoughts about her spouse. Similarly, if a man has the assumption that once a woman has been unfaithful in one relationship then she will always be so in others, it will result in thoughts of infidelity about his partner. The patient accepts these thoughts as true and frequently acts upon them.

Previous learning history, cultural and social factors and other influences such as personality help to form the schema in relation to sexual and emotional relationships. A salient precipitating factor will activate assumptions particularly related to the subjects' perceived self-efficacy within sexual relationships and their ability to maintain a relationship. Jealous thoughts concerning their partner's behaviour or intent will occur in response to external or internal stimuli (including other thoughts or mood states). Furthermore, similar to depression in which low mood increases the accessibility of negative cognitions, a negative view of self increases the accessibility of such thoughts.

In addition to cognitions and schemata, Beck (1983) hypothesised that each individual has a more stable personality structure which could function as 'a central value system of superordinate schemas'. This suggests that morbid jealousy patients should have different personality profiles than matched normals. Tarrier *et al* (1989) found that a sample of women who identified themselves as morbidly jealous scored significantly higher on introversion, neuroticism and psychoticism scales of the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975)

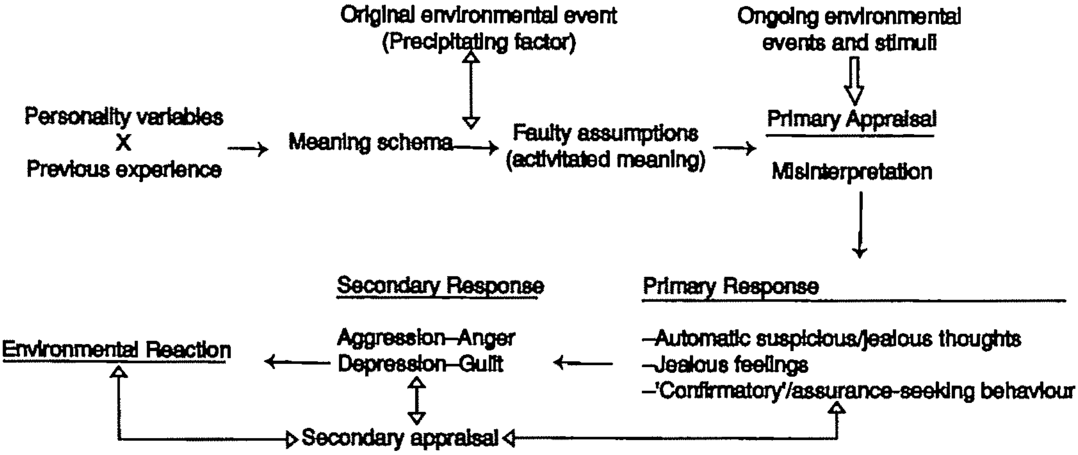


FIG. 1 Diagramatic representation of the processes which may underlie morbid jealousy.

than did an age matched normal population, but not on the lie score. The result on the neuroticism scale is especially pertinent as Bagley & Evan-Wong (1975) found a significant correlation between Eysenck's neuroticism trait and the 'negative evaluation of self'.

Fig. 1 presents a diagramatic representation of the processes which possibly underlie morbid jealousy. In this proposed model, jealousy is conceptualised as a primary response to an initial appraisal of environmental events. Such primary appraisals are based upon misinterpretations of events and reflect underlying schema, themselves formed by previous experiences and affected by personality variables. The misinterpretation of on-going events continues even though the original activating event has past. The nature of the activating event will vary between individuals and may be highly idiosyncratic.

Fig. 2 illustrates the development of morbid jealousy in a referred patient. In this example, the birth of the patient's first child activated a schemata which included beliefs about her personal attractiveness and the stability of her marital relationship. She assumed that the latter was dependent only upon her being sexually attractive to her husband. In a similar manner to depressed patients, the activated assumptions produce systematic distortions in the perception and interpretation of on-going events. The stimulus event of witnessing her partner in conversation with a female work colleague provokes the patient to evaluate herself as less attractive than the female colleague. The conversation

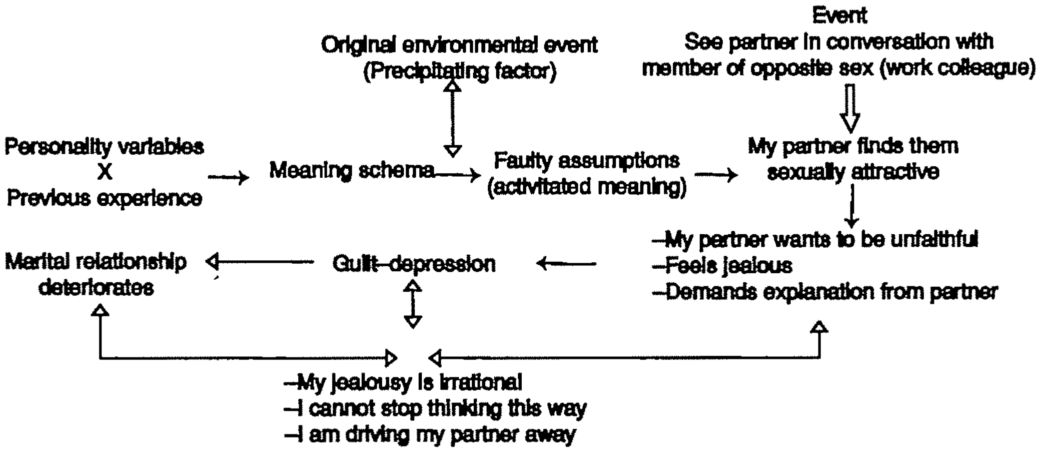

Event
See partner in conversation with member of opposite sex (work colleague)
Original environmental event
(Precipitating factor)
Personality variables
X
Previous experience
Meaning schema
Faulty assumptions (activitated meaning)
My partner finds them sexually attractive
–My partner wants to be unfaithful
–Feels jealous
–Demands explanation from partner
Marital relationship deteriorates
Guilt–depression
–My jealousy is irrational
–I cannot stop thinking this way
–I am driving my partner away

FIG. 2 Example of the development of morbid jealousy.

is misinterpreted as evidence that the spouse is sexually attracted to his female colleague. This results in automatic thoughts of suspicion accompanied by jealous feelings and the initiation of confirmatory behaviour.

In the morbidly jealous individual, faulty information-processing can result in a wide variety of events becoming triggers for further automatic suspicious thoughts, even in the absence of objective evidence. Magazine pictures, television programmes, social situations and variations in a partner's behaviour or routine may all be misinterpreted within the framework of a faulty assumption system and result in suspicious and jealous feelings. In this regard jealous thoughts share a number of characteristics with obsessional thoughts. They are intrusive, frequently unwanted, accessible, unpleasant and sometimes identified as irrational (if not at the time then subsequently). They are also frequently accompanied by behavioural actions such as checking or the seeking of reassurance. Jealous thoughts may also be considered as similar to intrusive thoughts, which are common and exist on a continuum from normal to pathological (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). Salkovskis (1985) in his cognitive–behavioural analysis of obsessions, suggested that ruminations are problematic because of the unpleasant automatic thoughts that occur in response to them. A similar situation can be hypothesised in morbid jealousy where an intrusive thought of suspicion or negative appraisal precipitates automatic thoughts of the partner's infidelity.

While morbid jealousy seems to be a primary response to the misinterpretation of events and stimuli, there appear to be secondary responses also (see Fig. 1). Such responses may range from avoidance behaviour, guilt and depression, to anger and aggression. Novaco (1979) in his cognitive–behavioural model of anger, conceptualises responses to external events as being determined by both primary and secondary appraisal processes (see also Chesney, 1985). In this model subsequent reactions are determined not only by the initial perception of events but also by the individual's coping style, effectiveness in coping, the anticipated outcome and the reaction of others. Thus, much of the behaviour, other than 'confirmatory', exhibited by jealous individuals may represent the outcome of secondary appraisal processes (Fig. 1). These responses are frequently characteristic of morbid jealousy, but are clearly not unique to it. Those individuals (see Fig. 2) who, on reflection, view their initial suspicion as irrational and their 'confirmatory' behaviour as unreasonable, are more likely to experience guilt and depression, and exhibit withdrawal, silence and behavioural avoidance in response to their jealousy.

With other individuals, however (see Fig. 3), secondary appraisal may serve to strengthen the initial beliefs. Such individuals will tend to accept their interpretation of the evidence as rational and view their 'confirmatory' behaviour as appropriate. In such circumstances, events which give rise to jealousy are likely to be experienced as provocative, and will result in anger and lead in some circumstances or individuals to aggression and violence.

Future research possibilities

At the present time there is little empirical data to support this model. We produced some support from

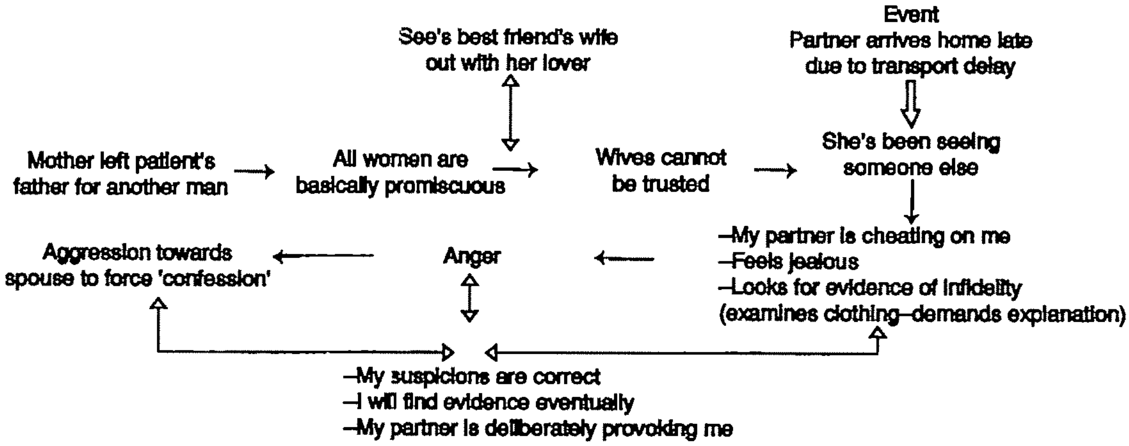

See's best friend's wife
out with her lover
Event
Partner arrives home late
due to transport delay
Mother left patient's
father for another man
All women are
basically promiscuous
Wives cannot
be trusted
She's been seeing
someone else
Aggression towards
spouse to force 'confession'
Anger
–My partner is cheating on me
–Feels jealous
–Looks for evidence of infidelity
(examines clothing–demands explanation)
–My suspicions are correct
–I will find evidence eventually
–My partner is deliberately provoking me

FIG. 3 Example of the development of morbid jealousy.

a successful but uncontrolled trial of cognitive therapy based on this formulation (Bishay *et al*, 1989). We also showed that morbidly jealous women differ from normal controls on a personality test (Tarrier *et al*, 1989) and we are presently analysing data from a survey of morbidly jealous individuals. However, the heuristic value of this formulation is that it may generate further investigation, and a number of research strategies suggest themselves. For example, studies on the relationship between mood and the accessibility of negative cognitions (Clark & Teasdale, 1982; 1985) may be useful in elucidating the relationship between emotional insecurity and jealous thoughts. Also, since it has been demonstrated, both in depressed patients (Derry & Kuipers, 1981) and in normal subjects with induced depressed mood (Sutton *et al*, 1988), that self-schema are predominantly organised to process self-related material negatively, similar experiments could be done to investigate morbid jealousy. Thus, by experimentally varying feelings of attractiveness, insecurity or self-esteem the accessibility of jealous thoughts would also be expected to change.

Another avenue which is potentially fruitful is to use methods from experimental psychology to investigate cognitive processing. Mathews & MacLeod (1985; 1986) and Watts *et al* (1986) have used a verbal reaction test, and Foa & McNally (1986) a dichotic listening task to demonstrate that fear-related words are processed differently in phobic and obsessional patients. Similar methods could be used to investigate the cognitive processing of infidelity-related words in jealousy patients.

Besides its theoretical interest, the utility of the cognitive–behavioural model is that it does suggest numerous treatment strategies. The systematic exposure of the patient to jealousy-provoking stimuli, the modification of dysfunctional cognitive processes, and the modification of antecedents such as poor self-esteem all merit investigation. There is also a pressing need for a refinement in the definition of problems and in the development of appropriate assessment measures and techniques. Finally, controlled treatment trials need to be carried out.

References

Abely, P. & Feuillet, C. (1941) Les idées delirantes de jalousie dans la demenoe prococe. *Annal Medical Psychologies*, 99, 75.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders.* (DSM–III) (3rd edn.) Washington, DC: APA.

Bagley, C. & Evan-Wong, L. (1975). Neuroticism and extroversion in response to Coopersmith's Self-Esteem Inventory. *Psychological Reports*, 36, 253–254.

Beck, A. T. (1967) *Depression: Clinical, Experimental and Theoretical Aspects*. New York: Haeber.

—— (1976) *Cognitive Therapy and Emotional Disorders*. New York: International Universities Press.

—— (1983) Cognitions, attitudes and personality dimensions in depression. *British Journal of Cognitive Psychotherapy*, 1, 1–16.

——, Rush, A. J., Shaw, B. F., *et al* (1979) *Cognitive Therapy for Depression*. New York: Guildford Press.

Bishay, N., Peterson, N. & Tarrier, N. (1989) An uncontrolled study of cognitive therapy for morbid jealousy. *British Journal of Psychiatry*, 154, 386–389.

Chesney, M. A. (1985) Anger and hostility: future implications. In *Anger and Hostility in Cardiovascular and Behavioural Disorders* (ed. M. A. Chesney & R. H. Rosenman). Washington: Hemisphere Press.

Chimbos, P. D. (1978) *Marital Violence: A Study of Interspouse Homicide*. San Francisco: R & E Research Associates.

Clark, D. M. & Teasdale, J. D. (1982) Diurnal variation in clinical depression and accessibility of memories of positive and negative experiences. *Journal of Abnormal Psychology*, 91, 87–95.

—— & —— (1985) Constraints on the effects of mood on memory. *Journal of Personality and Social Psychology*, 48, 1595–1608.

Cobb, J. (1979) Morbid jealousy. *British Journal of Hospital Medicine*, 21, 511–518.

—— & Marks, I. (1979) Morbid jealousy featuring as obsessive–compulsive neurosis. Treatment by behavioural psychotherapy. *British Journal of Psychiatry*, 134, 301–305.

Coen, S. J. (1987) Pathological jealousy. *International Journal of Psycho-Analysis*, 68, 99–108.

Daly, M., Wilson, M. & Weghorst, S. J. (1982) Male sexual jealousy. *Ethology and Sociobiology*, 3, 11–27.

Derry, P. A. & Kuipers, N. A. (1981) Schematic processing and self-reference in clinical depression. *Journal of Abnormal Psychology*, 90, 286–297.

Doherty, J. & Ellis, J. (1976) A new concept and finding in morbid jealousy. *American Journal of Psychiatry*, 133, 679–683

Enoch, M. D. & Trethowan, W. H. (1979) *Uncommon Psychiatric Syndromes* (2nd edn). Bristol: J. Wright.

Eysenck, H. J. & Eysenck, S. (1975) *Manual of the Eysenck Personality Questionnaire*. London: Hodder & Stoughton.

Foa, E. & McNally, R. J. (1986) Sensitivity to feared stimuli in obsessive–compulsives: a dichotic listening analysis. *Cognitive Therapy and Research*, 10, 477–485.

Gayford, J. J. (1975) Battered wives. *Medicine, Science and the Law*, 15, 235–245.

Gillies, H. (1976) Homicide in the West of Scotland. *British Journal of Psychiatry*, 128, 105–127.

Guttmacher, M. S. (1955) Criminal responsibility in certain homicide cases involving family members. In *Psychiatry and the Law* (eds P. H. Hoch & J. Zubin). New York: Grune & Stratton

Hafner, R. J. (1979) Agoraphobic women married to abnormally jealous men. *British Journal of Medical Psychology*, 52, 99–104.

Hilberman, E. & Munson, K. (1978) Sixty battered women, *Victimology*, 2, 460–470.

Im, W. G., Wilner, R. S. & Breit, M. (1933) Jealousy: interventions in couples therapy. *Family Process*, 22, 211–219.

Madden, J. S. (1979) *A Guide to Alcohol and Drug Dependence*. Bristol: Wright.

Mathews, A. & MacCloud, C. (1985) Selective processing of threat cues in anxiety states. *Behaviour Research and Therapy*, 23, 563–569.

—— & —— (1986) Discrimination of threat cues without awareness in anxiety states. *Journal of Abnormal Psychology*, 95, 131–138.

Mooney, H. (1965) Pathologic jealousy and psychochemotherapy. *British Journal of Psychiatry*, 111, 1023–1042.

Munro, A. (1984) Excellent response of pathologic jealousy to pimozide. *Canadian Medical Association Journal*, 131, 852–853.

Novaco, R. (1979) The cognitive regulation of anger and stress. In *Cognitive–Behavioural Interventions: Theory Research and Procedures* (eds S. D. Hollon & P. C. Kendall). New York: Academic Press.

Pollock, B. G. (1982) Successful treatment of pathological jealousy with pimozide. *Canadian Journal of Psychiatry*, **27**, 86–87.

Rachman, S. J. & de Silva, P. (1978) Abnormal and normal obsessions. *Behaviour Research and Therapy*, **16**, 233–248.

Rounsaville, B. J. (1978) Theories in marital violence: evidence from a study of battered women. *Victimology*, **3**, 11–31.

Salkovskis, P. M. (1985) Obsessional–compulsive problems: a cognitive–behavioural analysis. *Behavioural Research and Therapy*, **23**, 571–583.

Salkovskis, P. M. & Harrison, J. (1984) Abnormal and normal obsessions: a replication. *Behavior Research and Therapy*, **22**, 549–552.

Seeman, M. V. (1979) Pathological jealousy. *Psychiatry*, **42**, 351–358.

Shepherd, M. (1961) Morbid jealousy: some clinical and social aspects of a psychiatric syndrome. *Journal of Mental Science*, **107**, 687–704.

Shrestha, K., Rees, D. W., Rix, K. J. B., *et al* (1985) Sexual jealousy in alcoholics. *Acta Psychiatrica Scandinavica*, **72**, 283–290.

Stahly, G. B. (1978) A review of select literature of spousal violence. *Victimology*, **2**, 591–607.

Sutton, L. J., Teasdale, J. D. & Broadbent, D. E. (1988) Negative self-schema: the effects of induced depressed mood. *British Journal of Clinical Psychology*, **27**, 188–190.

Tarrier, N. Beckett, R. Harwood, S., *et al* (1989) Comparison of a morbidly jealous and a normal female population on the EPQ. *Personality and Individual Differences*, **10**, 1327–1328.

Teisman, M. W. (1979) Jealousy: systematic problem solving therapy with couples. *Family Process*, **18**, 151–160.

Watts, F., Frank, R., McKenna, F. P., *et al* (1986) Colour naming of phobia-related words. *British Journal of Psychology*, **77**, 97–108.

West, D. J. (1968) A note on murders in Manhattan. *Medicine, Science and the Law*, **8**, 249–255.

Wilt, G. M. (1974) *Towards an Understanding of the Social Realities of Participating in Homicide* (cited by Daly *et al*, 1982). Unpublished PhD Thesis, Wayne State University.

*Nicholas Tarrier, PhD, FBPsS, *Department of Psychology, Badham Clinic, University of Sydney, NSW 2006 Australia*; Richard Beckett, BSc, Dip Clin Psychol, *Department of Psychology, Prestwich Hospital, Salford Health Authority, Manchester M25 7BL, UK*; Susan Harwood, BA, *Department of Psychology, Prestwich Hospital*; Nagy Bishay, MRCPsych, *Department of Psychiatry, North Manchester General Hospital, Manchester, UK*

*Correspondence

Prothiaden

Dothiepin Hydrochloride B.P.

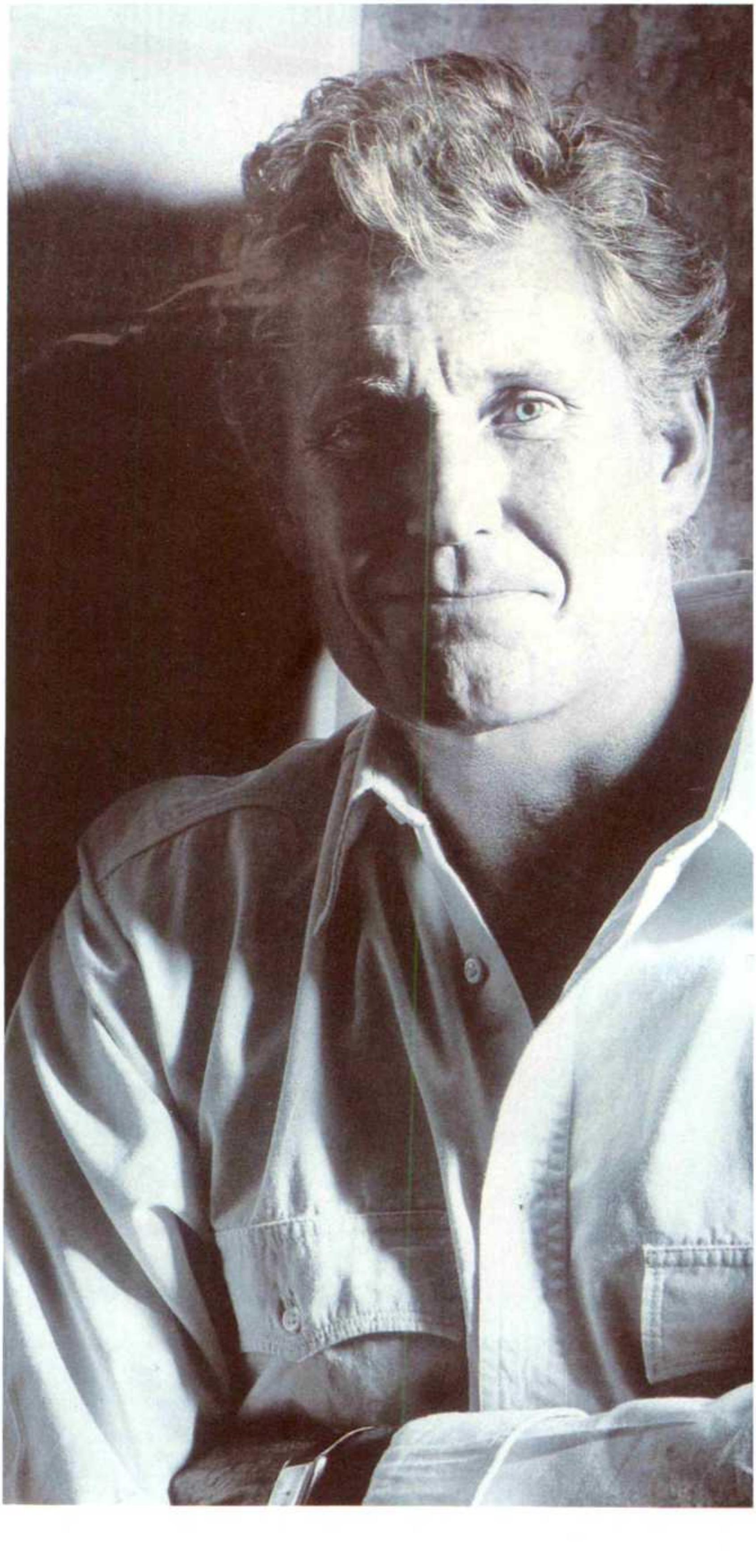
OOTS PHARMACEUTICALS

TOMORROW'S STANDARD TODAY

RESCRIBING INFORMATION Presentation: Prothiaden is Dothiepin Hydrochloride B.P. an antidepressant of the tricyclic group. It is available as 25mg capsules nd 75mg tablets. **Uses:** Prothiaden is indicated in the treatment of depression and associated anxiety. **Dosage:** Prothiaden should be given in a dosage of 75 to 150mg aily, based on response and severity. This may be taken as a single evening dose. In certain circumstances, i.e. in hospital use, Prothiaden has been given in dosages p to 225mg daily. Elderly – half the normal dose may be sufficient. **Precautions and Contra-indications:** Prothiaden has anticholinergic properties, therefore, it may recipitate urinary retention in susceptible individuals and its use should be avoided in patients with existing or potential urinary retention. Prothiaden should not be sed in patients with closed-angle glaucoma. Caution is advised when treating epileptic patients and those with cardiovascular disorders. Prothiaden should not be used vith MAO inhibitors nor within 14 days of ceasing such treatment. It may modify the action of some CNS depressants, catecholamines and hypotensive agents. **Side ffects:** These are usually mild and normally controlled by reducing the dosage. The following have been reported: dryness of mouth, constipation, disturbed ccommodation, orthostatic hypotension, palpitations, somnolence, tremor, headache. **Basic N.H.S. Price:** 28 × 75mg £4.00 as at 1st Oct. 1987 **Product Licence umbers:** PL0014/5941. PL0014/0209. Boots Pharmaceuticals, Nottingham, England. Further information available on request. ETH 1324/2/90

\BRIDGED PRODUCT
NFORMATION ▼ SPECIAL
REPORTING TO CSM REQUIRED
ndications: Acute and chronic
sychotic states. **Dosage: Adults:**
nitially 20-50mg/day in two doses.
ncrease over 7-10 days to 60-100mg/
lay in 2-4 doses. Usual maintenance
lose 20-100mg/day. Maximum daily
ose 250mg. **Children:** Not
ecommended. **Contraindications:**
Ise in comatose or semicomatose
atients or in severe drug-induced
epressed states (alcohol, barbiturates,
arcotics). Known hypersensitivity to
he drug. **Precautions:** May impair
nental/physical abilities — patients
hould be warned that the ability to
rive a car or operate machinery may be
ffected and about concomitant alcohol
r CNS depressants. Use with caution
n patients with:- history of convulsive
isorders, cardiovascular disease,
laucoma or a tendency to urinary
etention, pregnancy or lactation.
Varnings/Adverse Effects: Extra-
yramidal reactions have been reported
– reduction in dosage or anti-
arkinsonian medication may be
ecessary. Persistent tardive dyskinesia
nay appear after long-term therapy or
vhen drug therapy has been dis-
ontinued. The elderly or those on
igh dose therapy (especially females)
ppear to be most at risk of this
ide-effect. Dystonias have been
eported — dosage reduction, temporary
oxapine withdrawal or corrective
rugs may be required. Tachycardia,
ypotension, hypertension, light-
eadedness, syncope, dermatitis, facial
edema, pruritis, seborrhoea, rash and
nticholinergic effects have occurred.
Other effects reported have included
ausea, vomiting, weight-gain/loss,
yspnoea, ptosis, hyperpyrexia, flushed
acies, headache, paraesthesia, poly-
ypsia and rarely, neuroleptic malignant
yndrome. **Pregnancy & Lactation:**
afe use in pregnancy and lactation has
ot been established. **Drug Inter-**
ctions: Interactions with other CNS
epressant drugs, anti-parkinsonian
rugs and alcohol may occur. **Over-**
osage: Treatment is symptomatic
nd supportive. It should include
astric lavage and extended dialysis.
egal Category: POM. **Present-**
tions and Basic NHS Prices:
ottles of 100 Loxapac Capsules
ontaining Loxapine succinate 10mg
9.52, 25mg £19.05, 50mg £34.27.
icence Numbers: PL 0095/0036-
038. Further information is available
n request to the Company: Lederle
aboratories, Cyanamid House,
areham Road, Gosport, Hants PO13
AS. ®Loxapac is a trademark.
eferences:1. Paprocki J et al, Folha
led 1975; 70(6): 533-558. (5612) 2.
ernstein JG: Handbook of Drug
herapy in Psychiatry. Boston, John
Vright/PSG Inc, 1983: 92-93. 3.
aldessarini RJ, In: Goodman LS and
ilman AG (eds) The Pharmacological
asis of Therapeutics, 7th Edition.
ew York, Macmillan Publishing
ompany 1985: 387-405. 4. Bishop
P et al, Psychopharmacology 1977;
1: 107-115(5607). 5. Tuason VB
al, J. Clin Psychiatry 1984;
5:158-163(12749). 6. Martin A,
sychologie Medicale 1982; 14(9):
439-1452(11542). 7. Ayd FJ. Jnr, Int
rug Ther. Newsletter
976; 11(7): 25-28.

Lederle



Cool, calm and psychotic

Loxapac is a new class of antipsychotic agent which represents an important advance in the treatment of schizophrenia. Controlled double-blind studies have shown Loxapac to be as effective as haloperidol in acute schizophrenia[1], with less potential to cause troublesome extrapyramidal symptoms.[1-3].

Further controlled studies have shown Loxapac to be clinically superior to haloperidol[1], chlorpromazine[4,5] and trifluoperazine[4] in the treatment of paranoid schizophrenia. Even in patients refractory to other antipsychotic agents, Loxapac has demonstrated a high level of efficacy[6].

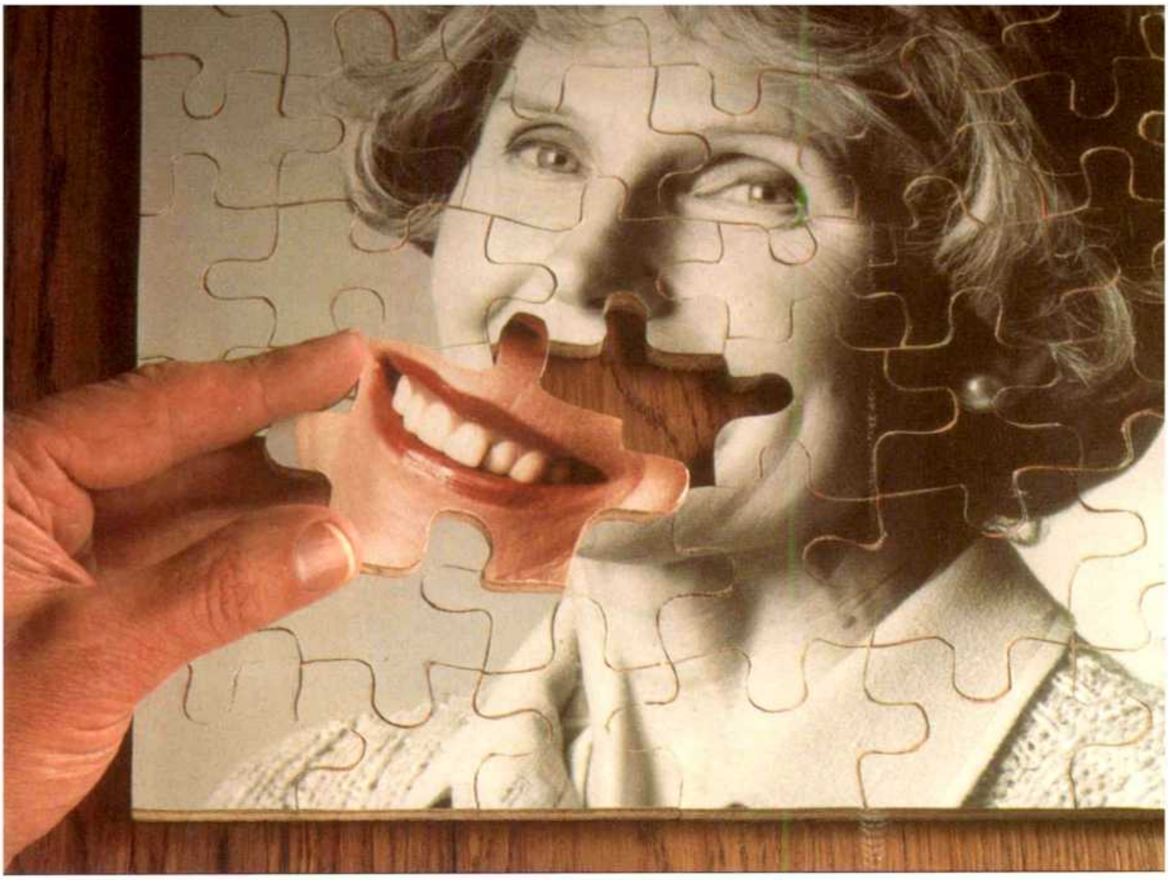
It has also been reported that Loxapac may suppress psychotic symptoms so well and so quickly that it may reduce hospitalisation time[7].

Thus if you want your psychotic patients to be calm and in control, choose new Loxapac to help them return to a normal, active life.

Loxapac

LOXAPINE

NEW ANTIPSYCHOTIC



Help put back what depression takes out

'averin is as effective as you might expect any
:-line antidepressant to be.

}ut Faverin is also generally non-sedating, so
er depressed patients are not affected by the
wsiness commonly associated with traditional
idepressants.

Jnlike tricyclics, Faverin produces few
icholinergic effects which can add to the
blems of ageing, like constipation and blurred
on.

Nor does Faverin produce any clinically
significant change in blood pressure or heart ra

And – importantly – there have been no deat
attributable to Faverin alone in intentional or
accidental overdosage up to 8600mg.

NEW SIMPLER DOSAGE

Faverin is now available in a new 100mg table
size, which means a simpler dosage regimen -
just one Faverin 100mg at night.

FAVERIN®

fluvoxamine ▼

ets the older depressed patient get on with li

:rin Prescribing Information ▼ Presentation: Round, yellow, enteric-coated
s each containing 50mg fluvoxamine maleate, imprinted with 'Duphar 291', in packs of 60
s, basic NHS price £25.00. PL 0512/0070. Oval, yellow, enteric-coated tablets each containing
g fluvoxamine maleate, imprinted with 'Duphar 313', in packs of 30 tablets, basic NHS price
0. PL 0512/0072. **Uses:** The treatment of symptoms of depressive illness. **Dosage and**
nistration: The tablets should be swallowed without chewing and with water. *Adults, including*
derly: Recommended starting dose of 100mg in the evening. Adjust according to response to
ıum of 300mg daily, in divided doses. *Children:* Not recommended. **Contra-indications,**
ings, etc: Avoid during pregnancy and in nursing mothers, unless compelling reasons. Do not
ıth, or within two weeks of ending treatment with monoamine-oxidase inhibitors. Faverin has

dose initially with careful monitoring. Discontinue if associated with increased hepatic enzy
effects of alcohol may be potentiated by Faverin. Improvement may be delayed for two or mc
and close monitoring is advised during this period. **Drug interactions:** Clinically si
interactions possible with drugs metabolised by liver and having narrow therapeutic i
warfarin, phenytoin and theophylline). Increased plasma levels of propranolol and warfar
practice. No interactions seen with atenolol or digoxin. **Side-effects:** Most c
gastrointestinal; nausea, vomiting and diarrhoea. In some patients who report nausea, vom
occur. Caution should therefore be exercised when administering Faverin to patients with a
condition likely to be exacerbated by vomiting. Others include dizziness, somnolence, a
headache and tremor. Anxiety reported less often. Convulsions have been reported. Avoid in

British Journal of Psychiatry (1990), 157, 327–330

Comment

Treatment of Alzheimer's Disease

Molecular Pathology Versus Neurotransmitter-Based Therapy

DAVID M. BOWEN

Cortical inhibitory neurotransmitters, neuropeptides, dopamine and noradrenaline are probably either not selectively or not critically affected in AD. It is, however, likely that a key change is shrinkage or loss of corticocortical pyramidal neurones, which probably use glutamate as their transmitter. This depletion appears to be circumscribed and clinically relevant.

Whalley (1989) has argued that treatment of Alzheimer's disease (AD) may be dependent upon understanding the "molecular pathology". One aspect, the formation and accumulation of neurofibrillary tangles within cortical pyramidal neurones, has been considered the major direct cause of nerve-cell degeneration. At present the excitement of the work on the structure of beta-amyloid protein in senile plaques, and the implications from knowledge of the cDNA sequence of its precursor, has overshadowed the tangle and pyramidal cell. For example, it has been suggested that understanding biochemical changes occurring after the amyloid precursor protein has been synthesised may eventually provide treatment (Caputo & Salama, 1989). The disease has also often been approached optimistically as purely a genetic disorder involving chromosome 21, yet recent research fails to establish this for families where the disease appears late in life (Pericak-Vance *et al*, 1988). It is generally accepted that there is a role for genes and amyloid-related proteins in pathogenesis but these are unlikely to be the only factors, so a broader view is necessary because it is likely that the clinical syndrome results from a series of different events over a long period (Bowen *et al*, 1988).

While AD is a slowly progressive disorder, this is rarely acknowledged in the interpretation of post-mortem biochemical studies; many neurotransmitters have been reported to be affected (Whalley, 1989), leading some to the pessimistic conclusion that a successful transmitter-based therapy will not evolve (Martin *et al*, 1988). It is argued herein, however, that dysfunction of the glutamatergic transmitter system associated with many pyramidal neurones is critical. This may be linked with perturbations of neuronal energy metabolism, a proposed pathogenic mechanism, which itself may be 'triggered' by an environmental factor, such as an infectious agent or exposure to aluminium (Bowen *et al*, 1988). Energy metabolism cannot be studied effectively with routine post-mortem material. The present commentary therefore lays emphasis on transmitters and indices of energy metabolism determined in neurosurgical samples obtained soon after the emergence of symptoms of the disease.

Preserved cortical transmitters

Brain tissue from sufferers of AD has been occasionally removed surgically for diagnostic purposes; a few samples have been offered to the author (39 from seven centres, between 1976 and 1987), and while the portions have been small, they seem to have been extremely useful (Fig. 1; Table I). GABA content as well as activity of its synthetic enzyme (GAD) and concentration of an associated peptide (somatostatin) were not selectively reduced ante-mortem (Fig. 1). Assessment post-mortem of GABAergic neurones has been complicated by artefact (Lowe *et al*, 1988). Thus, no change in GAD activity was found where AD and control subjects were carefully matched for agonal state (Reinikainen *et al*, 1988). Taurine is also thought to function as an inhibitory transmitter and the content was unchanged in AD (Lowe *et al*, 1990). Vasoactive intestinal polypeptide and neuropeptide Y were unaffected in post-mortem series, as were other peptides (cholecystokinin and galanin; Francis & Bowen, 1989) and possibly neurones containing corticotrophin-releasing factor (Kelley & Kowall, 1989). Indices of catecholaminergic neurones were in general not reduced in biopsy samples (Fig. 1). Of studies on dopamine-beta-hydroxylase activity post-mortem, one indicated reduced activity and the other found no change; dopamine content has consistently been found not to be reduced, and HVA concentrations were reported reduced in some regions, but elevated in others. Reduced concentrations of noradrenaline have generally been reported whereas concentrations of MHPG have been found to be reduced, unaltered, or even elevated (Francis & Bowen, 1989).

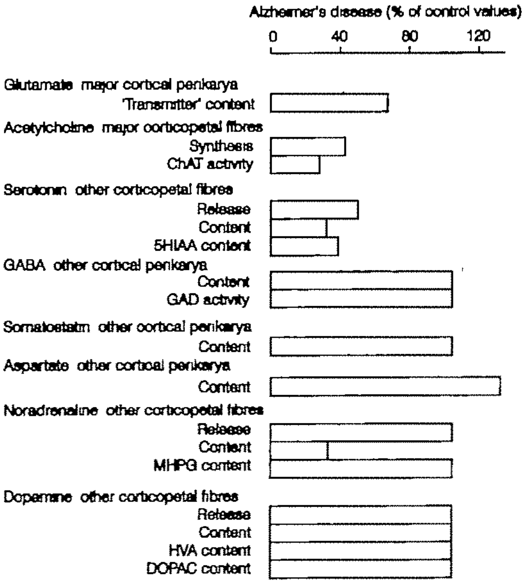


FIG.1 Selective neurotransmitter changes in ante-mortem (biopsy) samples of cerebral cortex from patients with AD. The cortex receives inputs from a number of subcortical neurones – four populations are shown here as corticopetal neurones. Non-significance is identified by 100% of control. GABA, gamma-aminobutyrate; GAD, glutamate decarboxylase; ChAT, choline acetyltransferase; 5-HIAA, 5-hydroxyindoleacetate; MHPG, 3-methyoxy-4-hydroxyphenylglycol; HVA, homovanillate; DOPAC, dihydroxyphenylacetate. (See Francis & Bowen, 1989; Lowe *et al*, 1990.)

TABLE I

The evidence for no hypometabolism in ante-mortem (biopsy) samples from patients with Alzheimer's disease

Tissue preparation	*Marker*	*Marker in samples from AD patients: % of control values*
Cerebral cortex homogenates	Oxygen uptake	
	+ADP (n=5,7)	120
	−ADP (n=5,7)	181*
	+CCCP (n=5,7)	102
Cerebral cortex tissue prisms	$[U\text{-}^{14}C]$glucose→$^{14}CO_2$	
	5 mMK$^+$ (n=9,17)	142**
	31 mMK$^+$ (n=22,27)	128**

ADP, adenosine diphosphate; CCCP, carbonyl cyanide m-chlorophenylhydrazone (an uncoupling agent). Five and 31 mMK$^+$ identify incubation conditions simulating resting and active neurones, respectively.

Values in parentheses are the number of independent samples studied for AD patients and controls, respectively.

*$P<0.05$; **$P<0.001$, significantly different from control. See Sims *et al* (1987), for review of the evidence.

Reduced glutamatergic transmission

Connections within the cerebral cortex, linking one area or group of cortical neurones to others, are known as corticocortical association fibres, and are thought to use an excitatory amino acid (EAA) as their transmitter. The cortex is usually divided into six layers; pyramidal neurones in layer III have axons forming mainly corticocortical fibres. In AD, degeneration of subsets of corticocortical association fibres is suggested by the distribution, seen by staining post-mortem brain, of tangles and senile plaques (Procter *et al*, 1988; Esiri *et al*, 1990). Loss of neurones giving rise to association fibres appears to occur relatively early in the disease, as the number of pyramidal cells was reduced in layer III of biopsy tissue from the temporal lobe (Neary *et al*,1986).

The temporal and parietal lobes may be major and early sites of pathological changes, as these regions showed apparent reduction in glucose metabolism by positron emission tomography (PET), a measurement in living brain which is sensitive to atrophy. Apparently contradictory results were obtained when cortical biopsy specimens were studied *in vitro* since no reduction in indices of energy metabolism was found (Table I). Neuropsychological test scores correlated with both PET data (Procter *et al*, 1989) and with the pyramidal cell counts in layer III (Neary *et al*, 1986). Thus, it is probable that a clinically relevant shrinkage or loss of association fibres occurred from certain circumscribed areas, and this contributed to an overall reduction in cerebral metabolism as assessed by PET.

Although pyramidal cells are probably the most abundant neuronal type in the mammalian neocortex, the transmitter(s) they use has not been established unequivocally. The best candidates are the EAAs, L-aspartate and L-glutamate. It is also a problem to separate terminals of cortical fibres arising from the cortical pyramidal cells from the other main source of excitatory terminals in the cortex, those of the input fibres from the thalamus. Thalamic pathology in the AD was, however, less prominent than were the cortical changes.

Cerebral cortex in general has glutamatergic nerve terminals, as there are both a Ca^{2+}-dependent, K$^+$-stimulated release of the EAAs and a high-affinity uptake system for the false EAA transmitter, D-aspartate (Peinado & Mora, 1986; Procter *et al*, 1988). D-aspartate uptake may be reliably measured in tissue from promptly performed autopsies and in such specimens it was reduced by 50% in the temporal cortex of AD patients (Procter *et al*, 1988), indicating that a large proportion of cortical glutamatergic terminals are in fact synapses of corticocortical fibres and that these are lost in AD. Glutamate content of biopsy samples was also reduced (Fig. 1) and the value for individual subjects was related to the density of pyramidal neurones in layer III (Lowe *et al*, 1990). The most straightforward explanation is that loss of corticocortical association fibres had influenced the glutamate content of these samples. In summary, an EAA, probably L-glutamate, appears to be the principal transmitter of the corticocortical association fibres that seem to degenerate quite early in AD.

Glutamate and excitotoxicity

Glutamate and abnormal energy metabolism have been linked with excitotoxicity in AD (Greenamyre & Young, 1989). Brain mitochondria oxidise substrates (normally derived from circulating glucose) when energy (in the form of adenosine triphosphate; ATP) is required, and normally the rate is tightly linked to ATP requirements, but if they perform oxidation without producing stoichiometric amounts of ATP, they are said to be uncoupled and convert substrates inefficiently into ATP. A widely documented change in AD was an apparent reduction in cerebral metabolism, based on the metabolic rate for glucose and oxygen, determined by PET, as well as blood flow. Table I, however, shows that the oxidative capacity of cerebral tissue from AD patients may actually be increased. This has been interpreted as reflecting a partial uncoupling of mitochondria (Sims *et al*, 1987), possibly within cell bodies of a subpopulation of cortical neurones. A consequence of altered energy metabolism might be that low concentrations of endogenous EAAs (e.g. glutamate) become toxic (Greenamyre & Young 1989): previously it was thought that EAA transmitters become neurotoxins only when present at excessive concentrations. Toxicity is thought to occur because EAAs act as agonists (i.e. they activate specific receptors). One such interaction helps to open a cation channel, thought to be a component of the membrane of pyramidal cells, and this could lead to intolerable increases in calcium concentration within these cells (Procter *et al*, 1989; Siesjo & Bengtsson, 1989). ATP is usually assessed in terms of the adenylate energy charge (ATP + ½ADP)/(ATP + ADP + AMP). The measure was not reduced in tissue prisms from AD patients incubated as in Table I, although the ATP content was significantly lower (21% reduction) (Sims *et al*, 1983).

The pyramidal neurones in the cortex are the cells that are particularly vulnerable in AD as they develop neurofibrillary tangles, degenerate, and may contain uncoupled mitochondria. Any of these abnormalities might index another event in the cortex that influences continued survival of some of the subcortical input cells (corticopetal neurones, Fig. 1). The event is at present hypothetical but may involve reduced output of trophic factors by subpopulations of cortical cells. Severe deficiencies in cholinergic and serotonergic inputs into the cortex clearly do occur early in the disease (Fig. 1) and the extent of both of these was related to the intensity of tangle formation in the cortex (Esiri *et al*, 1990; Francis & Bowen, 1989). The hypothesis that the pathology within the cortex 'drives' the changes in the input cells is more straightforward than one proposing that *two* separate subcortical regions are affected before changes occur in the cortex.

Further research is required to establish that a proportion of glutamatergic neurones degenerate because of excitotoxicity. One approach would be to show that either an antagonist of EAAs or manipulation of oxidative metabolism retards disease progression. Likewise, Caputo & Salama (1989) considered the development of a method for disrupting amyloid formation should be useful for evaluating the importance of the amyloidosis, but acknowledged that amyloid deposition may be considerably 'downstream' from the primary cause of AD.

Treatment of symptoms

Until the cause has been identified, it is sensible to attempt to treat the symptoms. There are no reports that directly relate an index of glutamate transmission to the magnitude of dementia, but there is evidence that EAA transmitter dysfunction relates to disturbances of memory and behaviour. First, histological and PET studies, reduced D-aspartate uptake, and loss of tissue glutamate all suggest that in AD a proportion of glutamatergic corticocortical neurones degenerate, and this is probably clinically relevant. Second, EAA transmission may normally be the chief factor that sustains the activity of corticocortical neurones in the cortex, where L-glutamate receptors and pyramidal cells are abundant and pyramidal cells are the synaptic targets of other pyramidal neurones. Third, dysfunction of glutamatergic neurones seem to be prominent in the hippocampus (Peinado & Mora, 1986; Procter *et al*, 1988, 1989), a structure critical for memory. Last, the best-characterised receptor for L-glutamate is associated with the cation channel described above; known as the N-methyl-D-aspartate (NMDA) receptor complex, animal studies suggest it is involved in neural pathways underlying memory and behaviour (McCabe & Horn, 1988).

Use of EAA agonists, such as glutamate, have been proposed as therapeutic agents, but these are potentially dangerous because of the possibility of drug-induced excitotoxic damage. Instead, manipulation of the glycine modulatory site of the NMDA receptor complex could be a more suitable way to restore the activity of the remaining pyramidal neurones. Compounds acting at this site that exhibit only partial agonist characteristics may be the most suitable (Greenamyre & Young, 1989); the antibiotic, D-cycloserine, is one example (Hood *et al*, 1989) and may provide a valuable tool to investigate this approach to treatment. Success would, of course, depend on many factors, including the extent to which the site is occupied by endogenous compounds, the magnitude of neuronal loss, and the functional state of glutamate receptors (Procter *et al*, 1989; Siesjo & Bengtsson, 1989) in cortex affected by AD.

Acknowledgements

The work of the author has been supported by the Alzheimer's Disease Society, Astra Alab, Brain Research Trust ("Miriam Marks Department") and the Medical Research Council.

References

Bowen, D. M., Beyreuther, K., Cross, A. J., *et al* (1988) Group report. Cell injury: molecular biology and genetic basis. In *Etiology of Dementia of Alzheimer's Type* (eds A. S. Henderson & J. H. Henderson), pp. 165–176. Chichester: John Wiley.

Caputo, C. B. & Salama, A. I. (1989) Authors' response to commentaries. *Neurobiology of Aging*, 10, 451–462.

Esiri, M. M., Pearson, R. C. A., Steele, J. E., *et al* (1990) A quantitative study of the neurofibrillary tangles and the choline acetyltransferase activity in the cerebral cortex and the amygdala in Alzheimer's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, 53, 161–165.

Francis, P. T. & Bowen, D. M. (1989) Tacrine, a drug with therapeutic potential for dementia: post-mortem biochemical evidence. *Canadian Journal of Neurological Science*, 16, 504–510.

Greenamyre, J. T. & Young, A. B. (1989) Author's response to commentaries. *Neurobiology of Aging*, 10, 618–620.

Hood, W. F., Compton, R. P. & Monahan, J. B. (1989) D-Cycloserine: a ligand for the N-methyl-D-aspartate coupled glycine receptor has partial agonist characteristics. *Neuroscience Letters*, 98, 91–95.

Kelley, M. & Kowall, N. (1989) Corticotropin-releasing factor immunoreactive neurons persist throughout the brain in Alzheimer's disease. *Brain Research*, 501, 392–396.

Lowe, S. L., Francis, P. T., Procter, A. W., *et al* (1988) Gamma-aminobutyric acid concentration in brain tissue at two stages of Alzheimer's disease. *Brain*, 111, 785–799.

——, Bowen, D. M. & Francis, P. T. (1990) Antemortem cerebral amino acid concentrations indicate selective degeneration of glutamate-enriched neurones in Alzheimer's disease. *Neuroscience* (in press).

Martin, J. B., Beal, M. F., Mazurek, M., *et al* (1988) Some observations on the significance of neurotransmitter changes in Alzheimer's disease. *Aging and the Brain* (ed. R. D. Terry), pp. 129–148. New York: Raven Press

McCabe, B. J. & Horn, G. (1988) Learning and memory; regional changes in N-methyl-D-aspartate receptors in the chick brain. *Proceedings of the National Academy of Science (USA)*, 85, 2849–2855.

Neary, D., Snowden, J. S., Mann, D. M. A., *et al* (1986) Alzheimer's disease: a correlative study. *Journal of Neurology, Neurosurgery and Psychiatry*, 49, 229–237.

Peinado, J. M. & Mora, F. (1986) Glutamic acid as putative transmitter of the interhemispheric corticocortical connections in the rat. *Journal of Neurochemistry*, 47, 1598–1603.

Pericak-Vance, M. A., Yamaoka, L. H., Haynes, C. S., *et al* (1988) Genetic linkage studies in Alzheimer's disease families. *Experimental Neurology*, 102, 271–279.

Procter, A. W., Palmer, A. M., Francis, P. T., *et al* (1988) Evidence of glutamatergic denervation and possible abnormal metabolism in Alzheimer's disease. *Journal of Neurochemistry*, 50, 790–802.

——, Wong, E. H. F., Stratmann, G. C., *et al* (1989) Reduced glycine stimulation of [^{3}H] MK801 binding in Alzheimer's disease. *Journal of Neurochemistry*, 53, 698–704.

Reinikainen, K. J., Paljarvi, L., Huuskonen, M., *et al* (1988) A postmortem study of noradrenergic serotonergic and GABAergic neurones in Alzheimer's disease. *Journal of the Neurological Sciences*, 84, 101–116.

Siesjo, B. K. & Bengtsson, F. (1989) Calcium fluxes, calcium antagonists, and calcium-related pathology in brain ischemia, hypoglycemia, and spreading depression; a unifying hypothesis. *Journal of Cerebral Blood Flow and Metabolism*, 9, 127–140.

Sims, N.R., Bowden, D. M., Neary, D. *et al* (1983) Metabolic processes in Alzheimer's disease: adenine nucleotide content and production of $^{14}CO_2$ from (U-^{14}C)glucose *in vitro* in human neocortex. *Journal of Neurochemistry*, 41, 1329–1334.

——, Finegan, J. M., Blass, J. P., *et al* (1987) Mitochondrial function in brain tissue in primary degenerative dementia. *Brain Research*, 436, 30–38.

Whalley, L. J. (1989) Drug treatments of dementia. *British Journal of Psychiatry*, 155, 595–611.

David M. Bowen, BSc, PhD, *Reader in Neurochemistry; Miriam Marks Department of Neurochemistry, Institute of Neurology (Queen Square), 1 Wakefield Street, London WC1N 1PJ*

British Journal of Psychiatry (1990), 157, 331–334

Stress and Puerperal Psychosis

I. F. BROCKINGTON, C. MARTIN, G. W. BROWN, D. GOLDBERG and F. MARGISON

Eighty-eight in-patients admitted to a psychiatric mother-and-baby unit and 80 randomly selected recently delivered women in the general population were interviewed using the LEDS. Only five of 33 patients (15%) with puerperal psychosis had provoking agents, which is less than the figure for women in the community (36%). Provoking agents were present in only 8 of 25 patients with post-natal depression, but they were present in seven of nine patients with pre-natal depression and eight of nine women in the community with pre-natal depression.

It has been long recognised that the puerperium is a time of high risk for mental illness (Esquirol, 1845; Marcé, 1858; Paffenbarger, 1961; Kendell *et al*, 1987). In the history of psychiatry, the concept of 'puerperal mania' or 'puerperal psychosis' was one of the first to emerge, and has recently been revived (Hamilton, 1962; Brockington *et al*, 1982). Latterly, other more minor post-natal disorders have been described, including the maternity blues syndrome, post-natal depression, and disturbances of the mother–infant relationship. The classification and definition of these different post-partum mental disorders is a matter of debate. At this point it seems worth distinguishing, at least provisionally, between three groups of illness severe enough to lead to admission to a psychiatric mother-and-baby unit:

(a) puerperal psychosis – mania, schizophrenia or schizoaffective disorder starting shortly after childbirth
(b) pre-natal depression – depression starting during pregnancy
(c) post-natal depression – depression starting post-natally.

In the search for the cause of these disorders, it is reasonable to investigate the social and psychological stress of childbirth. Clinical impressions suggest that there may be a difference between these three groups in their association with stressful events and difficult circumstances, with a relative lack of stress in puerperal psychosis. This hypothesis deserves to be examined empirically, with due attention to the difficulties of measuring 'stress'.

Having access to a large series of patients admitted to a mother-and-baby unit, we were in a position to test the hypothesis.

Methods

The Mothers-and-Babies Unit at the University Hospital of South Manchester (Withington Hospital) admits about 50 patients per year, 81% of whom come from South Manchester Health District and its neighbours (North and Central Manchester, Stockport, Tameside, Trafford and Salford). Over the study period (25 May 1979–31 May 1981) 113 patients were resident on the unit. Systematic observations were carried out on patients in the unit, and information was gathered from several information sources, which included a modified version of the Present State Examination (see Brockington *et al*, 1981), given to both patient and the nearest relative, and structured nursing observations.

A random sample of women giving birth in the Maternity Unit of the University Hospital of South Manchester was also taken. Ninety-two women were approached by letter six months after delivery, and were asked to take part in a study of women's experiences of childbirth and motherhood. All interviews were carried out at home. These mothers formed the sample for another study, reported in detail by Martin *et al* (1989), focusing on depression. Psychiatric state was assessed using the PSE in these subjects.

Diagnoses were made in terms of the Research Diagnostic Criteria (Spitzer *et al*, 1975) by two raters who reviewed all the available data. They worked independently at first, then reached consensus diagnoses.

In the community sample, diagnoses of depression were made by a team not involved in the social interviews, using the criteria of Finlay-Jones *et al* (1980).

Onset of illness was also determined by a review of all the available data. The patients and their relatives were questioned about the first symptom or sign of changed behaviour, or where the research data were incomplete, the case records were used. The onset was determined by two raters working independently, then reaching consensus. In line with previous experience (Brockington *et al*, 1982) puerperal psychosis was defined in terms of an illness starting within one month of delivery.

The use of the Life Events and Difficulties Schedule (LEDS) has been described elsewhere (Brown *et al*, 1973; Brown & Harris, 1978). The essence of the technique is that enough information is obtained to establish the presence of predefined types of events. In addition further information is collected about the person's past and present circumstances and behaviour in order to make an assessment of the likely threat of the event in terms of his/her ongoing concerns, plans, and purposes. The procedure is *contextual* in the sense that information about the person's actual responses to the event (and the presence of any psychiatric disorder) is ignored in making the rating. A somewhat similar procedure is followed for ongoing difficulties.

In this study, *events* rated as markedly or moderately threatening (the top two points of a four-point scale of severity and focused on the subject) and *difficulties* rated at the top three points of a six-point scale of severity (lasting two years and not involving health) were collectively referred to as 'provoking agents'. The time frame of the interview in the present study was 15 months, but the analysis was restricted to the 38 weeks before onset or interview. The research worker (CM) spent six weeks at Bedford College, London, learning the interview methods. She modified the schedule to include more detailed questions about pregnancy, childbirth and child-care. The ratings of contextual threat were made by members of the Bedford College team except for 11 patients, for whom they were made by CM. The reliability of her ratings when compared with the London team was high (kappa = 0.81).

Statistics

Chi-squared tests were used to compare the occurrence of life events and difficulties in the groups defined by diagnosis and timing.

Results

Of the 113 patients resident, 103 were approached, 13 declined interview, and two remained too ill to be interviewed; thus 88 were interviewed. Sixteen of these were not given the full programme of systematic clinical observations, because of discharge within five days (two patients), lack of current psychiatric illness (six patients), and other reasons, such as patient or consultant refusal or holidays (eight patients). Of the 92 women approached in the community, six refused interview and six had moved out of the area; thus 80 were interviewed.

Since the paper is concerned with puerperal psychosis and depression related to pregnancy, we excluded the following patients:

(a) four patients judged after admission to have no psychiatric illness; most were admitted for assessment of maternal competence
(b) 12 patients with non-depressive illness starting during pregnancy or before pregnancy (i.e. outside the puerperium as defined above) (mania, 4; schizophrenia or probable schizophrenia, 6; undiagnosed functional psychosis, 1; and 'other psychiatric disorder', 1)
(c) three patients with a diagnosis of schizophrenia with onset more than one month after delivery (i.e. outside the puerperium)
(d) two patients with chronic depression, excluded because it was impossible to determine the timing of events in terms of the onset of their depression (which was in any case before the beginning of pregnancy).

These exclusions leave 67 patients for the analysis. Their diagnoses can be broken down as follows:

(a) nine with pre-natal depression
(b) 33 with puerperal psychosis – eight with an early puerperal onset of mania or hypomania, six with schizoaffective mania, four with an illness with elements of both mania and depression, two with 'unspecified functional psychosis' (both of whom had some manic features), five with schizoaffective depression, and eight with schizophrenia or probable schizophrenia – all of whom had onset within four weeks of delivery
(c) 25 with post-natal onset of depression, of whom 17 had onset within one month of delivery.

In the community sample, two were depressed at the beginning of pregnancy, nine became depressed during pregnancy, and five after delivery.

Life events and difficulties

The number of patients in each group who reported recent severe events or major difficulties ('provoking agents') as defined above, is shown in Table I. In the community sample, 28 women had provoking agents in the 38 weeks before interview (36%).

Looking first at patients with severe stress as indicated by the combined presence of life events and difficulties, the differences between diagnostic groups are highly significant ($\chi^2 = 22.5$, 2 d.f., $P < 0.001$), and this was mainly due to the excess of severe stress in the patients with pre-natal depression. There was also a relative lack of such stress in the patients with puerperal psychosis.

If the presence of *either* a severe event *or* major difficulties is chosen as the index of stress, the differences are still highly significant, but less so ($\chi^2 = 13.3$, $P < 0.01$). Again the excess of events or difficulties in the patients with pre-natal depression made the greatest contribution, followed by the lack of events or difficulties in the patients with puerperal psychosis.

In the community sample, eight of the nine women with pre-natal onset of depression had a provoking agent, as did two of the five women with post-natal onset of depression and 18 of 64 without depression.

TABLE I
Provoking agents in patients admitted to a mother-and-baby unit

			Provoking agents	
Diagnostic group	n	*nil*	*event or difficulty*	*both event and difficulty*
Puerperal psychosis	33	28	5 (15%)	3 (9%)
Depression				
pre-natal onset	9	2	7 (78%)	7 (78%)
post-natal onset	25	17	8 (32%)	6 (24%)
Total	67	47	20 (30%)	16 (24%)

Discussion

Stress and puerperal psychosis

The object of this study was to test the hypothesis that puerperal psychosis is not associated with environmental stress. In the light of prior research, at least two forms of stress need to be distinguished. The first, severe long-term threat, represents the likely impact of pregnancy or birth on a woman's basic concerns and goals. Such threat is known to be capable of bringing about major depressive disorders (Brown & Harris, 1978). The second form of stress concerns an event of *any* type as defined by the LEDS, including events which only involve change in habit and routine, with little or no obvious threat. Events of this nature have been shown to accumulate in the three weeks before the onset of a florid schizophrenic episode (Brown & Birley, 1968). A birth is clearly an event in this sense. Given this, even if birth is unrelated to onset of a particular condition in terms of long-term threat, there is bound to be, in a study based on women giving birth, difficulty in ruling out an effect for such an event as a psychosocial rather than as a biological phenomenon.

The hypothesis that puerperal psychosis is not associated with environmental stress in terms of long-term threat has been confirmed by the present study, since only five of 33 patients with this diagnosis had a provoking agent during the previous 38 weeks. When this group of patients was compared with a sample of women from the general population (excluding the two who were already depressed at the onset of pregnancy), patients with puerperal psychosis had provoking agents less often (5/33, 15% v. 28/78, 36%), even than those not suffering from depression (18/64, 28%).

It would not be valid to make a statistical comparison between a non-random series of patients, subject to nosocomial bias, and a random community sample, but the difference is large and it is hard to explain why there should be *less* environmental stress in patients with this disorder than in the general population. One would expect, if anything, the presence of marked difficulties (e.g. lack of home support) to facilitate hospital admission, so that nosocomial bias would tend to exaggerate the rate for environmental difficulties in the patient series. The reasons for this difference are unknown, but it has not been shown to be statistically significant.

These findings, however, will come as no surprise to clinicians working with these patients, because it has long been recognised that this disorder has no obvious association with social disadvantage. Esquirol (1845) noted that it was common among the wealthy, and Gundry (1859) observed that "all classes seem equally liable. Neither riches, with the luxury that attends, nor poverty with its supposed exemption from enervation" protected against it. One index of social deprivation that has always been easy to ascertain is single parenthood; there is no consistent evidence that this is associated with puerperal psychosis, since the findings of several studies have produced conflicting results: Paffenbarger (1961) and Kendell *et al* (1976) found no association, but Tetlow (1955) and Kendell *et al* (1981) found a higher rate in unwed mothers. In Kendell's latest and largest study (Kendell *et al*, 1987) there was a significant association between the unmarried state and admission to hospital within one month of childbirth, since 19% of women admitted were unmarried, compared with 10% of mothers in the general population. The rate in patients with psychosis was also higher (with 16% unmarried), but this did not reach statistical significance. McNeil (1990) has recently completed a follow-up study of 88 women with a history of psychosis, and has found no association between post-partum psychosis and problems or lack of social support, assessed by interview during pregnancy.

Our study was, however, limited to the study of social stress, and does not provide any data on personality or pre-morbid functioning. The lack of environmental difficulties does not exclude the possibility that these patients had psychological difficulties, and the doctrine that this disease occurs in women with *normal personalities* living in normal circumstances remains a clinical impression.

Environmental stress in patients with depression related to pregnancy

Depression starting after childbirth was also associated with rather low rates of environmental stress. The proportion with a provoking agent was 32%, which is comparable to the frequency of such factors in the general population, although, as argued in the companion paper (Martin *et al*, 1989), provoking agents more commonly occurred in relation to the birth itself, or the first four weeks of birth, in those with post-natal depression than in healthy post-natal women in the general population and this difference reached statistical significance. However, the role of psychosocial stress in post-natal depression in the present series is at best modest and is less impressive than in some other research, which has reported an association with marital discord (reviewed by O'Hara & Zekoski, 1988) and recent stressful life events (Paykel *et al*, 1980).

We should, however, draw attention to a methodological problem which may have influenced our results to some extent. In our in-patient series there were four patients with a severe disturbance of the mother–infant relationship, who were rated as having 'no provoking agent' because the onset of the relationship disorder and the depression could not be dissociated. The relationship between post-partum depression and disorders of the mother–baby relationship is controversial, and methodological difficulties may have prevented us recognising the presence of this severe form of stress in a few of these patients. However, it is the marital rather than the maternal relationship which is generally regarded as disordered in post-natal depression, and the number of patients with mother–infant relationship disorders is not large enough to make a radical difference to our findings.

We found a strong association between pre-natal depression and social stress. Although numbers were small, this was true of both patient and community series. Depression with onset during pregnancy has not aroused the same interest as post-natal depression, but Paffenbarger (1961) noted the possible relationship of pre-natal mental illness to unwanted pregnancy, and this was confirmed by Kumar & Robson (1984). The association of pre-natal depression with severe stress merits further study.

Acknowledgements

We thank the consultants of University Hospital of South Manchester for permission to include their patients and the North West Western Regional Health Authority for financial support (locally organised project no. 358).

References

Brockington, I. F., Cernik, K. F., Schofield, E. M., *et al* (1981) Puerperal psychosis: phenomena and diagnosis. *Archives of General Psychiatry*, **38**, 829–833.

——, Winokur, G. & Dean, C. (1982) Puerperal psychosis. In *Motherhood and Mental Illness* (eds I. F. Brockington & R. Kumar), pp. 37–69. London: Academic Press.

Brown, G. W. & Birley, J. L. T. (1968) Crises and life changes and the onset of schizophrenia. *Journal of Health and Social Behaviour*, **9**, 203–214.

——, Sklair, F., Harris, T. O., *et al* (1973) Life events and psychiatric disorder: 1. Some methodological issues. *Psychological Medicine*, **3**, 74–87.

—— & Harris, T. (1978) *Social Origins of Depression*. London: Tavistock.

Esquirol, E. (1845) *Mental Maladies. A Treatise on Insanity* (trans. E. K. Hunt). Philadelphia: Lea & Blanchard.

Finlay-Jones, R., Brown, G. W., Duncan-Jones, P., *et al* (1980) Depression and anxiety in the community – replicating the diagnosis of a case. *Psychological Medicine*, **10**, 445–454.

Gundry, R. (1859) Observations on puerperal insanity. *American Journal of Insanity*, **16**, 294–320.

Hamilton, J. A. (1962) *Postpartum Psychiatric Problems*. St Louis: Mosby.

Kendell, R. E., Wainwright, S., Hailey, A., *et al* (1976) The influence of childbirth on psychiatric morbidity. *Psychological Medicine*, **11**, 342–350.

——, Rennie, D., Clarke, J. A., *et al* (1981) The social and obstetric correlates of psychiatric admission in the puerperium. *Psychological Medicine*, **1**, 341–350.

——, Chalmers, J. C. & Platz, D. (1987) Epidemiology of puerperal psychosis. *British Journal of Psychiatry*, **150**, 662–673.

Kumar, R. & Robson, K. (1984) A prospective study of emotional disorders in childbearing women. *British Journal of Psychiatry*, **144**, 35–47.

Marcé, L. V. (1858) *Traité de la Folles des Femmes Enceintes, des Nouvelles Accouchées et des Nourrices*. Paris: Baillière.

Martin, C. T., Brown, G. W., Goldberg, D. P., *et al* (1989) Psycho-social stress and puerperal depression. *Journal of Affective Disorders*, **16**, 283–293.

O'Hara, M. W. & Zekoski, E. M. (1988) Postpartum depression: a comprehensive review. In *Motherhood and Mental Illness* (eds R. Kumar & I. F. Brockington). Guildford: Butterworths

Paffenbarger, R. S., Jr (1961) The picture puzzle of the postpartum psychoses. *Journal of Chronic Diseases*, **13**, 161–173

Paykel, E. S., Emms, E. M., Fletcher, J., *et al* (1980) Life events and social support in puerperal depression. *British Journal of Psychiatry*, **136**, 339–346.

Spitzer, R., Endicott, J. & Robins, E. (1975) *Research Diagnostic Criteria*. New York: New York State Psychiatric Institute.

Tetlow, C. (1955) Psychoses of childbearing. *Journal of Mental Science*, **101**, 629–639.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *An Instruction Manual for the Present State Examination and CATEGO Programme*. Cambridge: Cambridge University Press.

*I. F. Brockington, MD, FRCP, *Professor of Psychiatry, Department of Psychiatry, Birmingham University, Queen Elizabeth Medical Centre, Birmingham B15 2TH*; C. Martin, PhD, *Research Fellow, Research Unit in Health and Behavioural Change, Edinburgh*; Professor G. W. Brown, FBA, *Department of Social Policy and Social Science, Royal Holloway and Bedford New College, University of London, 11 Bedford Square, London WC1B 3RA*; D. Goldberg, MD, FRCP, *Professor of Psychiatry, Department of Psychiatry, University Hospital of South Manchester, Manchester M20 8LR*; F. Margison, MSc, MRCPsych, *Consultant Psychotherapist, Central Manchester Health Authority, Gaskell House, Manchester*

*Correspondence

British Journal of Psychiatry (1990), **157**, 335–338

Is Psychiatric Training Still Improving?

PETER BROOK

A postal questionnaire inquiring about postgraduate training was directed to all consultants appointed to a post in general psychiatry for the first time between 1 October 1985 and 30 September 1986. Compared with previous surveys there is, overall, more expressed satisfaction with training in psychogeriatrics, supervised ward consultations, individual psychotherapy, work in the community, and with medico-legal experience. Diminished satisfaction is expressed in training in child and adolescent psychiatry, epidemiology, psychology, and neurology. An improved system of monitoring by the Royal College of Psychiatrists and the Joint Committee for Higher Psychiatric Training is suggested in order to further improve training.

From 1971 until 1982 all recently appointed consultants (NHS and honorary) who were working in general psychiatry and its associated specialities – psychogeriatrics, rehabilitation, and substance abuse – were asked their views as to how satisfactory their training had been. These postal inquiries, made at three-year intervals, eventually covered all general psychiatrists appointed between 1 October 1966 and 30 September 1981. The five cohorts will be referred to as the 1970, 1973, 1976, 1979, and 1982 groups (Brook, 1972, 1974, 1977, 1980, 1981, 1984). There was an initial improvement in expressed satisfaction with training compared with the results of an earlier, slightly different survey of consultants appointed during 1963–66 (Royal Medico-Psychological Association, 1969). There was little change until the 1979 survey, which did show some general improvement, a trend more clearly delineated for the 1982 group.

In the time covered by these surveys, two changes were introduced to improve postgraduate training. The Royal College of Psychiatrists, the successor to the Royal Medico-Psychological Association (RMPA), was founded in 1971. One of the College's first acts was to institute a membership examination, and over the next two years every hospital in the UK offering training was visited to assess its suitability for preparing young doctors for the examination. These visits have continued at regular intervals ever since. Guidelines for accreditation, based on those prepared by the RMPA, were formulated in order to help approval teams arrive at appropriate judgements. Over the years these standards have been progressively raised (Royal College of Psychiatrists, 1985).

The Joint Committee for Higher Psychiatric Training (JCHPT) was established in 1973 with its membership drawn from the main educational bodies in the specialty, the College and the Association of University Teachers in Psychiatry. The functions of the Committee were to publish recommended goals and standards of higher training in psychiatry in all its major specialties and to inspect and approve training programmes. Its broad aim was to ensure that those in higher training were being prepared adequately for consultant posts (JCHPT, 1987). Guidelines were published in the JCHPT's first report (1975) and inspection of schemes was in progress by 1976. In succeeding years standards required for schemes to be approved have been steadily raised.

This paper reports the experiences of a two-year cohort of recently appointed consultants.

Method

A postal questionnaire was sent to all 175 trainees appointed for the first time to a substantive post in general psychiatry (including those in special-interest jobs) in the UK between 1 October 1985 and 30 September 1986.

Respondents were asked to report their experiences in 13 basic clinical areas on a three-point scale:

1. adequate in quantity and adequate consultant supervision
2. adequate in quantity but inadequate consultant supervision
3. inadequate or no experience.

They were then asked to rate 12 areas of special experience or academic teaching on a similar scale:

1. sufficient in quantity, and well taught and properly supervised
2. sufficient in quantity, but inadequately taught and supervised
3. insufficient in quantity or none.

The wording of the two scales was as used in previous inquiries. The first 13 areas – identical to previous surveys except that the question on work in a therapeutic

TABLE I

Percentages of newly appointed consultants reporting complete satisfaction with training in clinical areas

Items on questionnaires			*1987*		
	total (n=*125)*	*trained in teaching hospital* (n=*66)*	*trained in non-teaching hospital* (n=*59)*	*UK graduates* (n=*103)*	*Overseas graduates* (n=*22)*
Interviewing techniques	54	59	49	54	55
Work with psychogeriatric patients	75[1]	74	78	80	59
Work with long-stay patients other than geriatric	54	52	56	56	41
Rehabilitation activities, e.g. occupational/ industrial therapy	35	35	36	39	18
Individual psychotherapy	72[1]	76	68	75	59
Group psychotherapy	58	59	58	61	46
Other methods of psychological treatment, e.g.:					
behaviour therapy, hypnotherapy	34	35	34	38	18
ward consultation in general hospitals	73[1]	73	73	72	77
treatment and management of the dependencies	61	59	63	58	73
emergency work (acutely disturbed patients in casualty depot or in patient's home)	67	70	64	68	64
medico-legal reports and sectioning	65[1]	64	66	66	59
Community work (including Part III accommodation, day centres and hospitals)	50[1]	50	51	50	55
Marital and family therapy	46	49	42	50	27

1. Notably greater than in the 1982 survey.

community was replaced by an inquiry about training in marital and family therapy – are listed in Table I. Table II gives the 12 areas of special experience; these are the same as in previous surveys except 'neurodiagnostic procedures' has been substituted for neuroanatomy and neurophysiology. In addition, respondents were asked to give their sex, and the medical school where they had obtained their original qualifications. They were asked to provide an outline curriculum vitae to determine the types of hospital where they had been trained.

As the results reflect opinion, formal statistical analysis was not considered to be appropriate.

TABLE II

Percentages of newly appointed consultants reporting complete satisfaction with teaching in specialised areas of experience

Items on questionnaires			*1987*		
	total (n=*125)*	*trained in teaching hospital* (n=*66)*	*trained in non-teaching hospital* (n=*59)*	*UK graduates* (n=*103)*	*Overseas graduates* (n=*22)*
Psychopharmacology	72	82	61	73	68
Mental handicap	29	29	29	30	23
Children and adolescents (not subnormal)	49[2]	52	46	54	23
Forensic (prison, remand home, approved school, borstal)	45	49	41	47	36
Neurology	46[2]	46	46	44	55
Psychology	53[2]	55	51	55	41
Epidemiology	41[2]	47	34	44	27
Administration	22	26	17	20	27
Teaching (undergraduate)	62	71	51	62	59
Teaching (postgraduate)	57	62	51	59	46
Research	55	59	51	52	73
Neurodiagnostic procedures	33	36	29	33	32

1. Notably greater than in the 1982 survey.
2. Notably less than in the 1982 survey.

Results

Replies were received from 145 doctors, of whom 20 proved to be ineligible; the final response rate of those falling within the scope of the survey was thus 81% (125/155), comparable with previous surveys. The present respondents are hereafter referred to as the 1987 group.

Twenty-two (18%) had qualified overseas, the great majority in the Indian subcontinent (1982 29%, 1979 26%, 1976 27%). Thirty-five (28%) were women (1982 19%, 1979 27%, 1976 20%).

As in the previous surveys, respondents were divided into two groups depending on the type of hospital (teaching i.e. university, hospital, or non-teaching) where they had received a majority of their training as a senior house officer or registrar.

Tables I and II give the 1987 consultants' ratings of their training and teaching compared with previous cohorts: only figures for the rating of adequate and adequately taught or supervised are given, as this is the standard for which training should be aiming. Less than half of the respondents reported complete satisfaction in: rehabilitation, other methods of psychological treatment, marital and family therapy, mental handicap, child and adolescent psychiatry, forensic psychiatry, neurology, epidemiology, administration, and neurodiagnostic procedures. The results of the previous surveys are presented in graphic form in Figs 1 and 2.

Discussion

Although there is a general trend towards improvement, progress apparently remains slow, posing questions about the Royal College's and the JCHPT's effectiveness in raising training standards. What must however be stressed is that all these surveys measured opinions only, so that what might have been regarded as satisfactory training experience ten years ago would now be seen as indifferent or even poor, owing to changing expectations because of the activities of the College and JCHPT. We are observing shifting standards as each cohort, especially recent ones, will have higher expectations as to what constitutes adequate teaching, thus blurring progress made as measured by the surveys.

As well as expectations, objective standards will also have been affected by the recommendations and inspections of the College and JCHPT. The standards of these inspecting and approving bodies have themselves risen over the last 15 years. A comparison of the JCHPT's first report (1975) with the most recent handbook (1987) makes it clear that recommendations have become more comprehensive, more detailed, and more precise. To take one example, a senior registrar who obtained a consultant post in 1988 at some time during training would have had at least six months' experience in child psychiatry together with formal lectures in that topic: 20 years ago very few newly appointed consultants would have had anything comparable in experience and training in child psychiatry. Examination of the figures indicates that it is since 1979 that most progress in training has taken place. Half to two-thirds of the doctors in the 1979 group would have started their psychiatric training in 1971–72 and in consequence would have benefited from the improved standards of training arising as a result of the efforts made by the JCHPT and the College. All those in subsequent cohorts would have had their training experiences shaped by the activities of these two bodies.

A further complicating factor is that consultant posts, especially in psychogeriatrics, have been expanding greatly in recent years but without a commensurate increase in the number of senior registrars. The consequence is that many senior

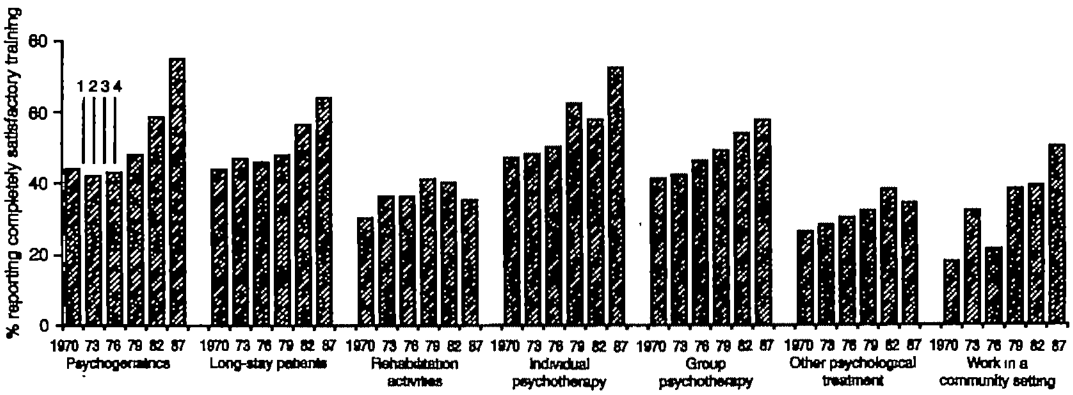


FIG. 1 Progress in psychiatric training in clinical areas, 1970–87. Note that in 1971 the College was established and in 1972 the membership examination and accreditation visits commenced; in 1975 the JCHPT published its guidelines, and in 1976 its approval visits started.

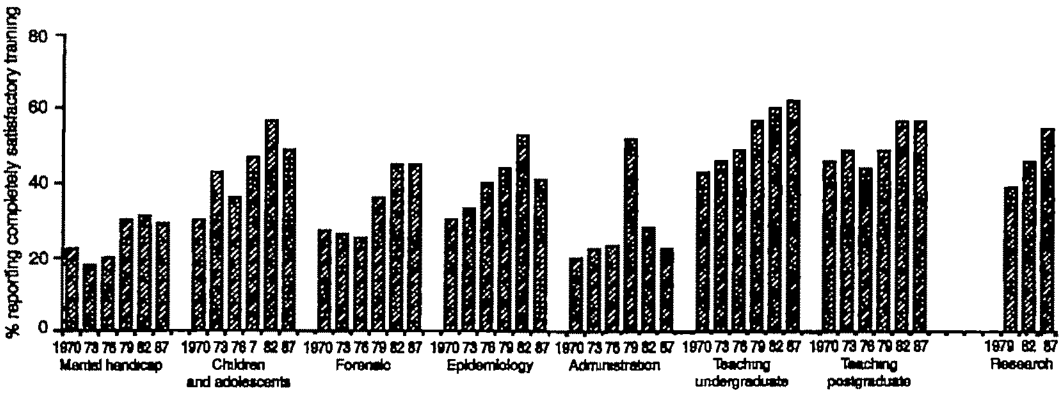


FIG. 2 Progress in psychiatric training in specialised areas, 1970–87.

registrars are appointed to consultant posts who have not completed a full training (Bewley, 1987).

Two final lessons can be drawn from the survey. The first is that there still exist differences between training in teaching (university) and non-teaching hospitals: these differences should disappear as more schemes incorporate rotation between both types of hospital, as a result of the recommendations made in *Plan for Action* (Department of Health and Social Security, 1988). The second is that there remains a continuing need for pressure by the College and the JCHPT to improve training standards generally and in certain areas specifically.

In order to achieve this aim the two organisations concerned need to improve monitoring of training and teaching. One way in which this could be done would be for trainees to rate their experiences at intervals, perhaps based on the questionnaire used in this inquiry. This would allow tutors and visiting teams to identify potential weak areas. Another way would be for senior registrars in conjunction with their tutors to identify any gaps or weaknesses while they were in training, and to set up 'learning contracts' to correct deficiences.

Acknowledgements

I would like to thank all those consultants who took part in the survey and Jane Boyce for her impeccable organisation of the survey. I am grateful to the DHSS, the Scottish Home and Health Department, and the DHS Northern Ireland for providing the names of consultants. Richard Wakeford, Senior Research Associate in the office of the Regius Professor of Physic, Cambridge, made helpful comments and suggestions. This project was funded by the Royal College of Psychiatrists.

References

BEWLEY, T. H. (1987) Joint Planning Advisory Committee. Report of meeting on 14 October 1986. *Bulletin of the Royal College of Psychiatrists*, 11, 72.

BROOK, P. (1972) Consultant psychiatrists: the background and training of recently appointed consultants. *British Journal of Medical Education*, 6, 190–195.

—— (1974) The postgraduate education and training of consultant psychiatrists. *British Journal of Psychiatry*, 124, 109–124.

—— (1977) Psychiatric training: the consultants' view. *British Medical Journal*, 1, 622–623.

—— (1980) Is psychiatric training improving? *British Medical Journal*, 281, 787.

—— (1981) Postgraduate psychiatric training: The Royal College of Psychiatrists' survey 1975–1978. *Medical Education*, 15, 335–339.

—— (1984) Consultant psychiatrists: their career choice and training. *Bulletin of the Royal College of Psychiatrists*, 8, 2–5.

DEPARTMENT OF HEALTH AND SOCIAL SECURITY (1988) *Hospital Medical Staffing: Achieving a Balance. "The Plan for Action". In Brief.* London: DHSS.

JOINT COMMITTEE FOR HIGHER PSYCHIATRIC TRAINING (1975) *First Report*. London: Royal College of Psychiatrists.

—— (1987) *Handbook*. London: Royal College of Psychiatrists.

ROYAL COLLEGE OF PSYCHIATRISTS (1985) Statement on approval of training schemes for general professional training for the M.R.C. Psych R.C. Psych. 1985. London: Royal College of Psychiatrists.

ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION (1969) Report on the questionnaire on postgraduate experience of training of consultants in adult psychiatry. *British Journal of Psychiatry*, 115, 225–231.

Peter Brook, MA, MD, FRCPsych, *Consultant Psychogeriatrician and Senior Registrar Organiser for East Anglia, Fulbourn Hospital, Cambridge CB1 5EF*

British Journal of Psychiatry (1990), **157**, 339–344

Psychiatric Diagnoses as Predictors of Suicide

A comparison of diagnoses at conscription and in psychiatric care in a cohort of 50 465 young men

PETER ALLEBECK and CHRISTER ALLGULANDER

In a cohort of 50 465 Swedish men conscripted for military service in 1969–70, the relative risk for suicide was 3.1 (95% CI 2.3–4.0) among those who had a psychiatric diagnosis at conscription, and 16.7 (95% CI 13.8–20.1) among those who had a psychiatric diagnosis in in-patient care during a 13-year follow-up. Of the diagnoses at conscription, only neurotic disorder, personality disorder and drug dependence were associated with a significantly increased risk for future suicide. Among those who were admitted to hospital, almost all in-patient diagnoses were associated with a significantly increased suicide risk. Although a psychiatric diagnosis in in-patient care was a strong predictor of suicide, only 44% of all 247 men who committed suicide had ever been treated in in-patient psychiatric care.

Psychiatric illness is one of the most powerful predictors of suicide (Balon, 1987; Hendin, 1986; Monk, 1987), and probably the most relevant one in the clinical context (Balon, 1987, Hawton, 1987). Although research on the relationship of demographic and social factors to psychiatric illness has helped to identify high-risk groups for suicide (Monk, 1987, Motto *et al*, 1985), the possibilities of prevention at the individual level are limited, because of the low specificity of any known predictor (Murphy, 1983; Balon, 1987; Hendin, 1986). Furthermore, the low base rate of suicide in the general population will give a low predictive value even for a test or a diagnosis which is strongly related to suicide (Murphy, 1983; Pokorny, 1983; Hawton, 1987). Nevertheless, several authors have pointed out that it is important to continue to provide clinicians with knowledge and instruments that can be helpful in the identification of subgroups with a particularly high risk of suicide (Hudgens, 1983; Hawton, 1987; Monk, 1987).

One of the problems regarding psychiatric illness as a predictor of suicide is that its predictive value depends on a number of factors: the population in which the diagnosis is made (hospital, out-patient clinic, general practice, etc.); when during the course of an illness the assessment is made; and the method of assessment (psychiatric interview, general screening interview, application of standardised scales). In this article we illustrate this problem using a longitudinal study in which psychiatric disorders were assessed at different points in time and in different settings.

In a previous article (Allebeck *et al*, 1988) we reported findings from a longitudinal study of 50 465 Swedish conscripts among which 247 suicides occurred during a 13-year follow-up. Psychiatric illness *per se*, assessed by a psychiatrist in the conscription survey, was not a significant predictor of suicide when compared with other baseline characteristics in a multivariate model. A specific diagnosis according to ICD–8 (World Health Organization, 1967) of neurotic disorder (ICD code 300) or personality disorder (301) was significantly associated with subsequent suicide, but a diagnosis of schizophrenic psychosis (295) or affective psychosis (296) was not. This cohort has now been followed in the national in-patient register for psychiatric care, which enabled us to analyse suicide rates associated with psychiatric diagnoses given in psychiatric care to members of the cohort. We could thus compare how future suicide is predicted by psychiatric diagnoses assessed in two very different types of setting.

Method

Conscription survey

During 1969–70, 50 465 men aged 18–20 years were conscripted for compulsory military training. They represented all Swedish men born between 1949 and 1951, except for 2–3% who were exempted, mainly because of severe handicap or congenital disorder. All men completed a questionnaire on family and social conditions and use of alcohol and narcotics. They subsequently underwent a psychological test that measured verbal, logical, inductive and technical ability. Each man had a structured interview with a trained psychologist, who then assessed him on a series of variables on the basis of data from the questionnaire, the test and the interview. Whenever a psychiatric disorder was reported by the conscript, or suspected by the psychologist, he was referred to a psychiatrist for assessment. In some cases the conscript had a certificate from the treating physician or the hospital, with a diagnosis or a brief case history, and a recommendation regarding the military placement. In other cases the psychiatrist could obtain further information from medical records, if the person had been treated. Any diagnosis made was coded according to the Swedish version of the ICD-8. During the years preceding this survey, efforts had been made to improve the quality of the psychiatric assessments (Otto, 1976).

The main purpose of the procedure was to place men within the military training scheme. Therefore, the threshold for referral to a psychiatrist was low, so that any psychiatric disorder would be detected early, and exemption from military training thus could be considered. The procedure is performed yearly, but only the data collected in 1969–70 were kept for research purposes, and were made available by permission of the research ethics committee of the Karolinska Institute and the Swedish Data Inspection Board.

Follow-up

The data from the conscription survey were linked to the national in-patient register for psychiatric care. This register covers all admissions to psychiatric clinics and mental hospitals in Sweden up to and including 1983, when the register was discontinued. All admissions were recorded with a personal number, unique to every citizen, a department identification, dates of admission and discharge and one or several diagnoses coded according to the ICD-8. The coverage of the register rose from 70% of all discharged cases in Sweden in 1969 to 83% of them in 1973, and was maintained at 97–98% during the remaining years of the follow-up.

Many patients had several diagnoses, and the order of the diagnoses (primary or secondary) could also differ between different hospitals. In particular, the diagnoses of alcohol and drug dependence were often associated with another psychiatric diagnosis. For this reason we calculated suicide rates by diagnosis irrespective of whether these were recorded as primary or secondary.

All deaths up to and including 1983 were identified by a linkage with the national cause-of-death register. This register covers death records for all deceased persons domiciled in Sweden. The causes of death are coded according to the ICD, from information on the death certificates. The necropsy rate in this cohort was 84%, and 83% of them were forensic necropsies.

Analysis

As in our previous study (Allebeck *et al*, 1988) we included the ICD categories 'suicide' (E950–959) and 'undetermined' (E980–989) in the analyses of suicide rates, in order to minimise any misclassification (Barraclough, 1973; Bolander & Ettlinger, 1980). The difference in suicide rate between those in each diagnostic group and those without a psychiatric diagnosis was calculated using a χ^2-test; in categories with less than ten suicides, a test based on the Poisson distribution was used. Relative risks were computed by dividing the suicide rate among those with a psychiatric diagnosis by the suicide rate among those without.

Results

Among 683 deaths from all causes, there were 247 deaths (36%) from suicide during the follow-up. Of these, 195 were definite suicides (E950–959) and 52 undetermined deaths (E980–989).

TABLE I

Number of suicides in conscripts with different psychiatric diagnoses

ICD-8 code	*Diagnosis*	*No. of conscripts with diagnosis*	*No. of suicides*	*Suicides per 100*	P[1]
292	Psychoses following intracranial infection	1	–	–	–
295	Schizophrenic psychoses	15	–	–	–
296	Affective psychoses	14	–	–	–
297	Paranoid states	2	–	–	–
298	Other psychoses	3	–	–	–
300	Neurotic disorders	2690	29	10.8	<0.001
301	Personality disorders	1442	22	15.3	<0.001
302	Sexual deviations	9	1	111.1	0.04
303	Alcohol dependence	218	4	18.3	0.02
304	Drug dependence	369	10	27.1	0.001
305	Psychosomatic disorders	485	2	4.1	0.6
306	Other special symptoms	625	5	8.0	0.2
307	Fortuitous psychic disorders	10	–	–	–
309	Mental disorders following somatic diseases	12	–	–	–
310–15	Mental retardation	619	5	8.1	0.2

1. From test comparing rate with that of conscripts without a psychiatric diagnosis.

TABLE II

Number of suicides in young men admitted to hospital during follow-up with different psychiatric diagnoses

ICD–8 code	*Diagnosis*	*No. of patients with diagnosis*	*No. of suicides*	*Suicides per 1000*	P^1
291	Alcoholic psychoses	76	1	13.2	0.5
292	Psychoses following intracranial infection	1	–	–	–
293	Psychoses following other nervous system disorder	16	1	62.5	0.1
294	Psychoses following somatic disease	33	3	90.9	0.003
295	Schizophrenic psychoses	304	25	82.2	<0.001
296	Affective psychoses	130	4	30.8	0.03
297	Paranoid states	29	3	103.4	0.002
298	Other psychoses	143	7	49.0	<0.001
299	Unspecified psychoses	200	13	65.0	<0.001
300	Neurotic disorders	895	48	53.6	<0.001
301	Personality disorders	513	27	52.6	<0.001
302	Sexual deviations	16	2	125.0	0.009
303	Alcohol dependence	1207	46	38.1	<0.001
304	Drug dependence	584	27	46.2	<0.001
305	Psychosomatic disorders	9	–	–	–
306	Other special symptoms	16	–	–	–
307	Fortuitous psychic disorders	92	7	76.1	<0.001
308	Abnormal childhood behaviour	1	–	–	–
309	Non-psychotic disorders following somatic disease	17	2	117.6	
310–15	Mental retardation	69	3	43.5	0.02

1. From test comparing rate with that of conscripts not admitted at follow-up.

TABLE III

Number of persons with a psychiatric diagnosis at conscription who were later admitted to hospital for psychiatric care, and number of suicides among those who were admitted to hospital

ICD–8 code	*Diagnosis*	*No. of conscripts with diagnosis*	*No. of persons admitted*	*(%)*	*No. of suicides among those admitted*
292	Psychoses following intra-cranial infection	1	–	–	–
295	Schizophrenic psychoses	15	7	(47)	–
296	Affective psychoses	14	7	(50)	–
297	Paranoid states	2	–	–	–
298	Other psychoses	3	2	(67)	–
300	Neurotic disorders	2690	366	(14)	18
301	Personality disorders	1442	261	(18)	12
302	Sexual deviations	9	2	(22)	1
303	Alcohol dependence	218	74	(34)	2
304	Drug dependence	369	127	(34)	6
305	Psychosomatic disorders	485	48	(10)	1
306	Other special symptoms	625	52	(8)	2
307	Fortuitous psychic disorders	10	1	(10)	–
309	Mental disorders following somatic diseases	12	–	–	–
310–15	Mental retardation	619	112	(18)	4

Of all 247 suicides, 71 occurred among those who had a psychiatric diagnosis at conscription and 108 among those with a diagnosis during in-patient care. This represents a relative risk for suicide of 3.1 (95% confidence intervals 2.3–4.0) among those with a diagnosis at conscription and 16.7 (95% confidence intervals 13.8–20.1) among those with a diagnosis during in-patient care.

Table I shows the suicide rates associated with different diagnoses at the time of conscription. Besides sexual deviations (all homosexuals), with only one suicide, drug and alcohol dependence were associated with the highest suicide rates. Of the other diagnostic groups, only neurotic disorders and personality disorders were associated with significantly increased suicide rates.

Table II shows the suicide rates associated with different diagnoses in in-patient care during the follow-up. Almost all diagnoses were associated with significantly increased suicide rates. Again, sexual deviations (homosexuality) were associated with the highest suicide rate, although there were only a few cases. Also, paranoid states had a high suicide rate, again with few cases. Patients with psychotic disorders in general had higher suicide rates than those with neurotic disorders, personality disorders and alcohol and drug dependence. It should be noted that these latter four diagnostic categories accounted for around 75% of all diagnoses. This proportion remained the same if only primary diagnoses were included.

The extent to which people with a psychiatric diagnosis at conscription were later admitted to hospital for psychiatric care is shown in Table III. Only 14% of those with a diagnosis of neurotic disorder and 18% of those with a personality disorder were admitted. The proportion of those admitted was higher for most other diagnostic categories.

Fifty per cent of those with a diagnosis of neurotic disorder at conscription were given the same diagnosis during hospital care. The remaining 50% had a variety of diagnoses, the most important ones being personality disorders, alcohol and drug dependence and schizophrenic disorders. A total of 35% of those with a diagnosis of personality disorder at conscription had the same diagnosis when in hospital care. Again, the other diagnoses given were alcohol and drug dependence, neurotic disorders and schizophrenic disorders.

Comparing the numbers of suicides in Tables I and III it can be seen that in most diagnostic categories, only 50–60% of those with a psychiatric diagnosis at conscription and who later committed suicide had been admitted to hospital. The major diagnoses given in hospital care, among these suicide cases who had been admitted, were as follows: drug dependence (28%); unspecified psychoses (ICD code 299; 20%), neurotic disorders (16%), alcoholic dependence (10%), schizophrenic disorders (9%) and personality disorders (8%).

Discussion

Our main finding was that the risk for suicide was much higher among those with a diagnosis in in-patient care than among those who were diagnosed in a screening of a total population. There were also substantial differences in suicide rates by type of diagnosis in the two settings.

This can be explained by several factors, one being that patients who are admitted to hospital generally have more severe psychiatric disorders than those who are not. Similarly, certain conditions, such as schizophrenia and affective psychosis, carry a higher likelihood of admission than others like personality and psychosomatic disorders.

The effects of time and age may also explain the differences in suicide risk. This is illustrated by the different patterns of diagnoses (Tables I and II). Schizophrenia and other psychotic disorders were not common at 18–19 years of age, but developed during the follow-up period up to 35 years of age. Conversely, disorders manifested as neurotic or personality disorders at conscription might have either disappeared over time, or developed into other disorders, such as substance abuse or psychosomatic disorders. We could also show that only a minority of those with a diagnosis of neurotic disorder or personality disorder at conscription had these same diagnoses in subsequent hospital care.

A third explanation is the method of diagnosis. The conscription examination is a screening procedure, in which most diagnoses are made after one or a few interviews, with the main purpose being to assess the suitability for military training. In contrast, diagnoses in in-patient care are made after thorough investigation, often over a considerable length of time, and the aim is to give adequate treatment. Given these differences, it might rather seem surprising that there was an overall agreement of around 50% in the diagnoses made in the two settings. Although there is good cause for scepticism regarding the validity of the diagnosis made in the conscription, they were made by trained psychiatrists, after initial screening by psychologists, who had been given special training for the purpose. Furthermore, the character of the procedure is most relevant for the question under study, namely to what extent a psychiatric diagnosis made in a screening situation is a useful predictor of future suicide. Any such screening of a large population would suffer from similar quality problems.

Monk (1987, p. 57) reviewed five large follow-up studies in which it was possible to determine the risk of suicide in a cohort of people with psychiatric illness and in the population from which the cohort originated. The studies by Pokorny (1983), Black *et al* (1985), Barner-Rasmussen *et al* (1986), and Babigian & Odoroff (1969), based on patients diagnosed in hospital care, showed an excess risk for suicide among psychiatric patients, ranging from 8.0

to 26.8 in men and 11.13 to 41.3 in women. Our finding of a relative risk for suicide of 16.7 (95% 13.8–20.1) among admitted psychiatric patients is thus in agreement with these studies. The lower risk for suicide among those with a psychiatric diagnosis at conscription in our study (3.1, 95% CI 2.3–4.0) is, however, at variance with the results of Hagnell *et al*'s (1981), although both studies were based on surveys of the general population.

In Hagnell *et al*'s (1981) general survey of psychiatric illness in Lundby, Sweden, the relative risk for suicide among those men in the survey who had a mental disorder (regardless of treatment) was 38.6 compared with those with no mental disorder. This study was based on only 23 suicides, and random variation might explain the discrepancy. Furthermore, as Monk (1987) pointed out, it is difficult to compare the results from different cohort studies, since they are based on different lengths of follow-up, different criteria for admission to psychiatric care and different completeness of follow-up. It is nevertheless possible that the discrepancy depends on the differences in sensitivity of the screening procedures. The relative risk for suicide among those without any mental disorder was 0.12 compared with those in the total Lundby cohort (Hagnell *et al*, 1981), indicating that almost all who later committed suicide were classified as having a mental disorder.

Our findings are in agreement with many studies pointing out psychiatric illness as the most powerful predictor of suicide. Miles (1977) estimated suicide rates associated with different psychiatric diagnoses, based on a review of over 100 follow-up studies, and concluded that the majority of suicides are associated with a small number of conditions. The studies reviewed were based on heterogeneous materials, and there are few follow-up studies that have analysed suicide rates for different diagnoses in one cohort. In our cohort of young men, schizophrenia and substance abuse were associated with higher suicide rates than indicated by Miles (1977) and others (Hendin, 1986; Monk, 1987). The suicide rate among those with affective psychosis was, however, lower than expected. It should be noted though that affective psychosis is a diagnosis which was not often made in young men during the time of the follow-up. Neurotic depression (300.40) was a much more common diagnosis (Akiskal *et al*, 1978), and of all those with a diagnosis of neurosis at conscription, 24% (655/2690) had depressive neurosis, and of all those admitted to hospital with neurosis, 50% (449/895) had depressive neurosis. It is thus natural that neurosis would be associated with a high suicide rate. Using the DSM–III diagnostic criteria Kovacs *et al* (1984) showed that adolescents with a dysthymic disorders (largely corresponding to neurotic depression) run a high risk of developing a major depressive disorder (corresponding to affective psychosis) in subsequent years and that these disorders are strongly inter-related in a longitudinal perspective.

Although the predictive value for suicide of a psychiatric diagnosis among conscripts was much lower than in cases admitted for treatment, the suicide rates based on diagnoses at conscription are not without interest. They call for general preventive measures in collaboration with social services and treatment units where persons with antisocial behaviour, and dependence on alcohol and drugs, are more frequent than in psychiatric care.

Consistent with the conclusions made by Murphy (1983) and Pokorny (1983), the suicide rate was substantially increased only in those men in the cohort who were admitted to psychiatric care during the follow-up. Only in patients who have previously attempted suicide has the excess risk for suicide been found to be even higher (Hawton & Fagg, 1988). It should be pointed out, however, that only 44% of those who committed suicide in our cohort had ever been admitted to psychiatric care. This illustrates the point made by Kreitman (1982) that very large cohorts are needed to analyse suicide rates even for the most vulnerable groups, such as psychiatric patients.

Acknowledgements

The project was supported by a grant from the Swedish Medical Research Council, no B89-27X-08322-02. We thank Lena Brandt for skilful computer programming.

References

Akiskal, H. S., Bitar, A. H., Puzantian, V. R., *et al* (1978) The nosological status of neurotic depression: a prospective three- to four-year follow-up examination in the light of the primary–secondary and unipolar–bipolar distinction. *Archives of General Psychiatry*, 35, 756–766.

Allebeck, P., Allgulander, C. & Fisher, L. D. (1988) Predictors of completed suicide in a cohort of 50 465 young men: role of personality and deviant behaviour. *British Medical Journal*, 297, 176–178.

Babigian, H. M. & Odoroff, C. L. (1969) The mortality experience of a population with psychiatric illness. *American Journal of Psychiatry*, 126, 470–480.

Balon, R. (1987) Suicide: can we predict it? *Comprehensive Psychiatry*, 28, 236–241.

Barner-Rasmussen, P., Dupont, A. L. & Bille, H. (1986) Suicide in psychiatric patients in Denmark, 1971–81. 1. Demographic and diagnostic description. *Acta Psychiatrica Scandinavica*, 73, 441–448.

Barraclough, B. M. (1973) Differences between national suicide rates. *British Journal of Psychiatry*, 122, 95–96.

Black, D. W., Warrack, G. & Winokur, G. (1985) Excess mortality among psychiatric patients. *Journal of the American Medical Association*, 253, 58–61.

Bolander, A.-M. & Ettlinger, R. (1980) Suicide in Sweden. In *Health and Society. V. Different Aspects of Mortality in Sweden*, pp. 39–72. Stockholm: Committee for Future-Oriented Research. Forskningsrådsnamnden.

Hagnell, O., Lanke, J. & Rorsman, B. (1981) Suicide rates in the Lundby study: mental illness as a risk factor for suicide. *Neuropsychobiology*, **7**, 248–253.

Hawton, K. (1987) Assessment of suicide risk. *British Journal of Psychiatry*, **150**, 145–153.

—— & Fagg, J. (1988) Suicide, and other causes of death, following attempted suicide. *British Journal of Psychiatry*, **152**, 359–366.

Hendin, H. (1986) Suicide: a review of new directions in research. *Hospital and Community Psychiatry*, **37**, 148–154.

Hudgens, R. W. (1983) Preventing suicide *New England Journal of Medicine*, **308**, 897–898.

Kovacs, M., Feinberg, T. L., Crouse-Novak, M., *et al* (1984) Depressive disorders in childhood: II. A longitudinal study of the risk for a subsequent major depression. *Archives of General Psychiatry*, **41**, 643–649.

Kreitman, N. (1982) How useful is the prediction of suicide following parasuicide? In *New Trends in Suicide Prevention* (eds J. Wilmotte & J. Mendlewicz). Basel: Karger.

Monk, M. (1987) Epidemiology of suicide. *Epidemiologic reviews*, **9**, 51–69.

Miles, C P. (1977) Conditions predisposing to suicide: a review. *Journal of Nervous and Mental Disease*, **164**, 231–246.

Motto, J. A., Heilbron, D. C. & Juster, R. P. (1985) Development of a clinical instrument to estimate suicide risk. *American Journal of Psychiatry*, **142**, 680–686.

Murphy, G. W. (1983) On suicide prediction and prevention. *Archives of General Psychiatry*, **40**, 343–344.

Otto, U. (1976) Male youths. A sociopsychiatric study of a total annual population of Swedish adolescent boys. *Acta Psychiatrica Scandinavica* (suppl.) **264**, 7–23.

Pokorny, A. D. (1983) Prediction of suicide in psychiatric patients. *Archives of General Psychiatry*, **40**, 249–257.

World Health Organization (1967) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Eighth Revision of the International Classification of Diseases* (ICD–8). Geneva: WHO.

*Peter Allebeck, MD, PhD, *Associate Professor of Social Medicine Department of Medicine, Section for Community Medicine, Karolinska Institute at Huddinge University Hospital, S-14186, Huddinge, Sweden*; Christer Allgulander, MD, PhD, *Associate Professor of Psychiatry, Department of Psychiatry, Karolinska Institute at Huddinge University Hospital, Sweden*

*Correspondence

British Journal of Psychiatry (1990), 157, 345–350

Schizophrenia and Crime
A Longitudinal Follow-up of 644 Schizophrenics in Stockholm

PER LINDQVIST and PETER ALLEBECK

To compare the crime rate of schizophrenics with that of the general population, data from the Central Swedish Police Register on 790 schizophrenic patients discharged from hospitals in Stockholm in 1971 was analysed for the period 1972–86. Relative risk of criminal offence was assessed by comparing the observed number of offences committed with the expected number (estimated from official statistics). The crime rate among male schizophrenics was almost the same as that in the general male population, whereas among females it was twice that of the general female population. The rate of violent offences was, however, four times higher among the schizophrenics. The violence recorded was almost exclusively of minor severity.

There is some uneasiness concerning the increasing crime rates in Sweden (Wikström, 1985), as well as the ongoing de-institutionalisation of the mentally ill. The role of psychiatry in predicting and preventing criminal behaviour among the mentally ill has also been questioned (Christie, 1982).

Few authors have investigated the general pattern of crime among the mentally ill compared with that of 'normal' subjects. Eitinger (1978) concluded from a study of 179 mentally ill offenders in Norway that there are two main categories of offenders: those who commit violent acts in acute, psychotic paranoid states in response to particular situations, and those who commit minor criminal offences to the same extent as an equivalent underprivileged non-psychotic population. Wiersma (1966), on the basis of a study in the Netherlands, claimed that individuals with schizophrenia contribute very little to crime in general, and Monahan & Steadman (1983) in the USA found that 'true' rates of crime are independent of mental disorder when allowing for demographic factors and certain life-history factors.

Using a population-based register of criminal offences, we were able to follow longitudinally a cohort of patients with schizophrenia, discharged from hospitals in Stockholm in 1971. To our knowledge, there has previously been no such study of crime among schizophrenics or other mentally ill people. The aims of the present study were:

(1) to find out if patients with schizophrenia commit crimes more frequently than the general population, and if so, to what extent;
(2) to describe the types of crime committed by schizophrenic patients, compared with the general crime pattern in Sweden.

Method

Study population

From the Stockholm county in-patient register we identified all patients ($n = 790$) born between 1920 and 1959, and with a diagnosis of schizophrenia (diagnostic code 295 according to ICD-8; World Health Organization, 1967) who were discharged during 1971. The register covers all psychiatric clinics and mental hospitals in Stockholm county, and thus provided a population-based cohort of schizophrenics for this area. The selection procedure, as well as a 10-year mortality follow-up, has been described previously (Allebeck & Wistedt, 1986). For the purposes of the present study, death records up to the end of 1985 were obtained, giving a total number of 142 deaths in the cohort during the 14-year follow-up period. These individuals had to be excluded from the study since information on deceased are effaced from the register of criminal records.

The validity of the diagnosis in the original cohort was assessed by Kristjansson *et al* (1987), who found that at least 85% of the cases complied with the DSM-III (American Psychiatric Association, 1980) criteria for schizophrenia. Four individuals identified in the present study were excluded since their medical records revealed psychosis other than schizophrenia. The number of schizophrenics studied here was thus 644.

Criminal records

The Swedish Central Police Board has a special register, The Central Police Register, which contains information on verdicts as well as on cases which are not prosecuted because of, for example, age or mental abnormality (*The Swedish Code of Judicial Procedure*, 1968).

Records of the register are kept for at least five years for offenders who are fined, and a minimum of ten years if a more severe sentence is given. Information is available from 1945 onwards in cases of repetitive criminal behaviour and felonies. Offences of *The Penal Code of Sweden* (1972) which are restricted to punishment by fines are not registered.

Follow-up and statistical analysis

Since the in-patient register and the Central Swedish Police Register are organised in terms of personal numbers, unique to every citizen, the records of both registers could be automatically linked at an individual level. Permission for this was granted by the Data Inspection Board and the Faculty Research Ethics Committee, University of Umeå.

Between 1972 and 1986, the Central Swedish Police Register was thus searched for information about the 644 schizophrenic patients. Only offences resulting in a verdict or a decision not to prosecute because of mental abnormality were included. Non-violent offences were analysed only for the period 1976–1986 since these crimes – in contrast to violent offences – more often result merely in fine penalties and are thus stored in the register for a shorter period of time.

The criminal activity of schizophrenics was compared with that of the general population using the number of 'principal offences' committed. The 'principal offence' for each person is that committed during one year which is linked to the most severe penalty. The number of principal offences in the two groups was analysed by indirect standardisation (Armitage, 1987), using the sex-specific and age-specific (10-year interval) rates of offence in the general Swedish population. The expected number of principal offences was obtained by multiplying the number of years of follow-up of each patient in the cohort by the yearly incidence of principal offences in the national population (Yearbook of Legal Statistics, 1980). The expected number of offences thus obtained among the schizophrenics was then compared with the observed number by computing the ratio of the observed : expected number of offences. The figure thus obtained corresponds to the relative risk of criminal offence among schizophrenics compared with the general population. Confidence intervals of 95% were calculated according to the test-based method described by Miettinen (1985).

Non-prosecuted danger-stamped psychiatric patients ('0-cases')

The term '0-cases' is applied to psychiatric in-patients who commit an act directed against the personal safety of another, but for which no prosecution is carried out. Classification as an '0-case' is determined by the chief psychiatrist at a hospital and/or the discharge committee and results in restrictions of leave and discharge. According to the statutes, most crimes against other people (physical or verbal), sexual offences, robbery and arson should be included as potential '0-cases' (Ekblom, 1970). However, in practice, the more severe violence by in-patients is generally reported to the police for investigation and prosecution. All '0-cases' are meant to be registered in the Central Swedish Police Register. Twelve such cases were identified, but omitted from the study since they were not judicially categorised. They will, however, be analysed in a future paper, where the violent offenders of the cohort are further examined.

TABLE I

Sex and age distribution of 644 patients with schizophrenia, discharged from hospitals in Stockholm county during 1971

Age: years[1]	*Sex*		*Total*
	Males n *(%)*	*Females* n *(%)*	n
12–20	15 (60)	10 (40)	25
21–29	121 (62)	75 (38)	196
30–39	96 (47)	109 (53)	205
40–51	98 (45)	120 (55)	218
Total	330 (51)	314 (49)	644

1. Age on January 1 1972.

Results

Incidence of criminal offence

The age and sex distribution of the 644 individuals is shown in Table I. Among these, 45 men and 9 women were responsible for 164 valid indictments between 1976–1986. Table II shows the relative risk of criminal offence among the schizophrenics compared with that of the general population. The crime rate among women was approximately twice the expected value whereas among men it was virtually the same as that of the general male population.

Figure 1 shows, for example, that in the age group 50–59 years, the number of principal offences committed by female schizophrenics was similar to the expected number in the female standard population. However, male schizophrenics in this age group had a crime rate that was more than twice that in the general male population.

Recidivism, defined as reconviction within five years (Yearbook of Legal Statistics, 1980), was similar among the schizophrenic men (22%), and the general male population (25%). Female schizophrenics had almost a doubled recidivism rate (23%) as compared with the general female population (14%).

Types of crime

Violent offences

Four of the 32 violent offenders were women (13%). The female contribution in national figures of corresponding crimes was between 5 and 10%. None of the women in the cohort committed two or more violent offences during the follow-up period, whereas seven men received two verdicts of violence, and three men were guilty of three violent offences each.

Assault, unlawful threat and threat or violence against officials constituted 95% of all violent crimes. Assault represented the most common offence and occurred in 20 instances (Table III). The most serious crime recorded was a charge of aggravated assault.

The group studied in the present investigation committed almost four times as many violent offences as the general

TABLE II

Observed and expected numbers of any and violent principal offences[1] (crimes) among 644 patients with schizophrenia

	No. of observed cases	*No. of expected cases*	*Ratio observed/ expected cases*	*95% confidence interval*
Any crime (1976–1986)				
men	75	69.5	1.2	0.7–2.1
women	17	7.3	2.2	1.5–3.6
Violent crimes (1972–1986) total	43	11.0	3.9	3.0–5.1
against life and health	20	7.4	2.7	1.8–4.1
against liberty and peace	8	1.4	5.7	3.1–10.5
against public order	15	2.3	6.5	4.2–10.0

1. Principal offence – the offence during one year which is linked to the most severe penalty.

population, this varying with the type of crime and age (Table II; Fig. 2). However, statistical comparisons between these subgroups must be interpreted with caution, and calculations regarding sex distribution versus type of crime was not performed because of the small numbers.

Non-violent offences

Table IV shows the main crime categories and the most frequent offences committed. Five women and 42 men were responsible for 131 valid indictments. Theft, petty theft, fraud and fraudulent conduct constituted half of all instances of non-violent crimes. Included in the group 'other offences' were four crimes of alcohol or drug use (drunk and disorderly behaviour, driving intemperated, drunken driving, breaking the Narcotic Drug Law), and three crimes against morals (carnal abuse of children and indecent conduct).

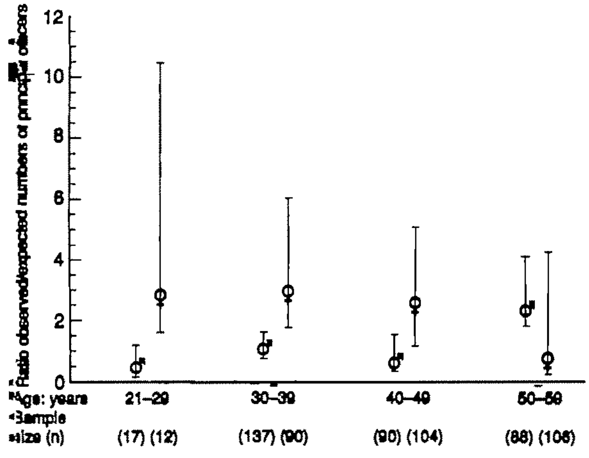


FIG. 1 Sex and age distribution of the ratio of observed : expected numbers of principal offences among 644 schizophrenics, 1976–1986 (age and sample size of the cohort taken from January 1 1980). Mean values of ratio shown by ♀ (women) and ♂ (men); 95% confidence interval marked by vertical lines.

TABLE III

Classification and number of violent offences among 644 patients with schizophrenia

Crime typology with corresponding chapter (and passage) in the Penal Code of Sweden	*No. of principal offences[1]*
Chapter 3 (1–11§): crimes against life and health	
aggravated assault	1
assault	15
petty assault	4
Chapter 4 (1–5§): crimes against liberty and peace	
unlawful deprivation of liberty	1
unlawful threat	7
Chapter 17 (1,16§): crimes against public order	
violence against officials	6
violence against officials (petty)	5
attempt of violence against officials (petty)	1
threat against officials	3
All violent crimes	43

1. Principal offence – the offence during one year which is linked to the most severe penalty.

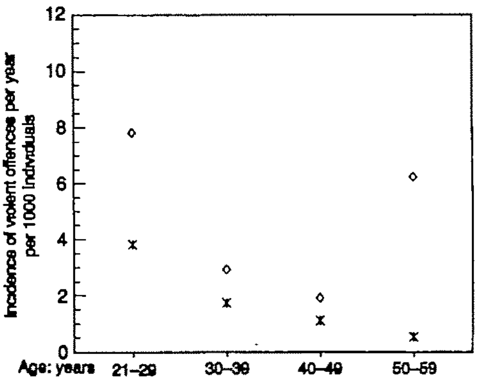


FIG. 2. The age-specific incidence per 1000 people per year (1972–1985) of the 42 schizophrenic patients (◇) who were responsible for assault, unlawful threat, and threat or violence against officials compared with the age-specific incidence of violence in offences in the general population (*) in 1978.

TABLE IV
Classification and number of non-violent offences among 644 patients with schizophrenia

Crime typology with corresponding chapter (and passage) in the Penal Code of Sweden	*No. of valid indictments*
Chapter 8: theft, robbery and other crimes of stealing	
(1§) theft	12
(2§) petty theft	24
(5§) robbery	1
Chapter 9: fraud and other dishonesty	
(1§) fraud	11
(2§) fraudulent conduct	18
Chapter 12: crimes inflicting damage	
(1§) inflicting damage	13
Chapter 17: crimes against public order	
(4§) violent resistance	6
Other offences	46
All non-violent crimes	131

Discussion

The main results of this study were that male schizophrenics did not, in general, commit more criminal offences than that expected in the general male population. The age distribution of male schizophrenic offenders was almost inverted as compared with findings in a study of custodially remanded male schizophrenic prisoners from the London area (Taylor & Parrott, 1988). However, different selection procedures and the small subgroups of this series may well be responsible for this discrepancy. Female schizophrenics had a criminal rate twice that of the general female population, although the actual numbers were small.

Violent crimes were four times more frequent among schizophrenics than 'normals'. This finding supports the conclusions of a study on remanded prisoners in England, in which schizophrenics were reported to have an increased risk of committing violent offences (Taylor & Gunn, 1984).

We have found no previous study with such a design that permits strict comparison with our findings, although a number of authors have investigated the question of dangerousness among the mentally ill as a group (e.g. Rappeport & Lassen, 1965; Rubin, 1972; Lagos *et al*, 1977; Teplin, 1985) or among specific psychiatric categories (e.g. Benezech *et al*, 1980; Craig, 1982, Häfner & Böker, 1982). Some methodological problems of such studies have been scrutinised by Krakowski *et al* (1986). In another critical review of 12 American papers on the subject, the author asserted "... we must be circumspect in imputing any special degree of criminality to the discharged mental patient" (Cohen, 1980). Nevertheless, several authors have reported that paranoid schizophrenia is correlated with an increased risk of violent behaviour (Tardiff & Sweillam, 1980; Addad *et al*, 1981; Krakowski *et al*, 1986). However, it is not self-evident that a mental illness *per se* creates socially illegitimate behaviour (Taylor, 1982), although it has been argued that overt psychotic symptoms are associated with risk for violent conduct (Rossi *et al*, 1986). Personality traits may also be a relevant factor for crime (Blackburn, 1968; Virkkunen, 1974*b*); the role of alcohol in aggressive behaviour is still disputed (Collins, 1982; Heath, 1983); and situational factors, including the often ambiguous interaction between the offender and the victim, are also not to be neglected (Wolfgang, 1958; Virkkunen 1974*a*,*b*; Planansky & Johnston, 1977).

Psychiatric personnel seem to perceive dangerousness in a systematic and rational manner (Segal *et al*, 1988; Mills, 1988). However, if discharged mental patients are denied appropriate social and medical support, criminal behaviour may ensue (Cohen, 1980). De-institutionalisation of the mentally ill therefore may have repercussions in terms of increase in violence by discharged and neglected mental patients (Gottlieb *et al*, 1987; Tanay, 1987; Weller & Weller, 1988).

From Swedish official statistics (1980) it was found that 14 000 individuals were sentenced to imprisonment while 800 were sentenced to social or psychiatric treatment. Thus, most criminals have few, if any, connections with psychiatry, and it is therefore dubious whether psychiatrists should accept the role of being society's safeguards against deviant behaviour, especially as 'normal' criminals are released without prior risk evaluation.

Society's legal actions against potentially dangerous individuals is not primarily a statistical or medical issue, but an ethical one. Two contradictory positions are recognised: the right of a mentally ill person not to be classed as a 'false positive' (Steadman, 1980), and the right of the public to protect itself against possibly harmful individuals (Walker, 1978).

The validity of the study

Since some socially well-adapted schizophrenics may well live independently of in-patient clinics, the in-patients studied here do not completely represent the schizophrenic population in this area. However, most schizophrenics are admitted to hospital at some stage during the course of their illness, and scrutiny of medical records showed that the patients in this cohort were in fact outside institutions during most of the follow-up time. A possible crime-reducing effect of staying in hospital, a result of supervision and medication, may influence the incidence of crime among mentally ill people (Binder *et al*, 1988). On the other hand, the intense and involuntary interaction in a psychiatric institution is liable to generate conflicts and unlawful behaviour (Ekblom, 1970).

The '0-cases' present a problem, since they could not be included in the analysis. However, the general population must have included some criminals who acted violently in custody, but do not appear on the register. Thus, although we might have missed some assaults committed by schizophrenics, the effects are at least partly balanced by a similar loss in the comparison group.

Since the Central Swedish Police Register does not cover all single and minor offences for more than five years, nor all crimes connected with more severe penalties than fines for more than ten years, some uncertainty remains as to what extent all offences are covered in our study. However, the crimes possibly missing are very minor ones and the numbers of reported cases and the ratio of solved cases can be assumed to be even lower among these crime categories than in the case of serious offences. Nevertheless, there is still uncertainty about how far the true incidence of petty offences is reflected in the official statistics.

In summary, there are factors that lead to an over-reporting as well as an under-reporting of crime among the mentally ill. On balance, we do not believe that these uncertainties would lead to a systematic bias in one direction, which might invalidate our findings.

We did not statistically adjust for residency and socio-economic status, since such information was not available in the records. However, we did find that arrest rate of violent offenders in Stockholm (i.e. where our study was based) was 1.9 times higher than that of the country as a whole. We might thus have overestimated the excess crime among schizophrenics, since our comparison group probably had a lower general crime rate than a corresponding group in Stockholm.

As regards social factors, it is well known that chronically ill patients with impaired community functioning tend to end up among the lower socio-economic strata of the population where criminal behaviour is more prevalent. If this is part of the disease pattern, it is doubtful whether it should be controlled for in the analysis. This could mask the real effects of mental illness, of which 'social drift' is only one aspect.

In spite of these shortcomings, we do feel that a population-based follow-up of a diagnostically homogeneous group of patients, which identifies offences through links with a police register, is valuable in describing the pattern of criminal offences of the mentally ill, and in quantifying the problem as compared with the general population.

Conclusions

The main findings of our study were that the rate of criminal offence among male schizophrenics was, in general, almost the same as in the general population, whereas among females it was twice that of the general population. The rate of violent offence was, however, four times higher among schizophrenics, but taking into account the problems of validity discussed above, this figure might be slightly overestimated. As in the general population, males dominate as offenders. Although the rate of offences among schizophrenics thus seems higher than in the general population, the ethical dilemma remains: how many and which schizophrenic patients should be retained on the basis of possible future criminal conduct?

Acknowledgements

This work was supported by the Bank of Sweden Tercentenary Foundation. The authors also acknowledge the expert secretarial assistance of Ms Lolomai Órnehult.

References

Addad, M., Benezech, M., Bourgeois, M., *et al* (1981) Criminal acts among schizophrenics in French mental hospitals. *Journal of Nervous and Mental Disease*, 169, 289–293.

Allebeck, P. & Wistedt, B. (1986) Mortality in schizophrenia. *Archives of General Psychiatry*, 43, 650–653.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (DSM–III) (3rd edn). Washington, DC: APA.

Armitage, P. (1987) *Statistical Methods in Medical Research*. Oxford: Blackwell Scientific.

Benezech, M., Bourgeois, M. & Yesavage, J. (1980) Violence in the mentally ill: a study of 547 patients at a French hospital for the criminally insane. *Journal of Nervous and Mental Disease*, 168, 698–700.

Binder, R. L. & McNiel, D. E. (1988) Effects of diagnosis and context on dangerousness. *American Journal of Psychiatry*, 145, 728–732.

Blackburn, R. (1968) Emotionality, extraversion and aggression in paranoid and nonparanoid schizophrenic offenders. *British Journal of Psychiatry*, 115, 1301–1302.

Christie, N. (1982) *Limits to pain*. Oxford, UK: Martin Robertson & Company.

Cohen, C. I. (1980) Crime among mental patients – a critical analysis. *Psychiatric Quarterly*, 52, 100–107.

Collins, J. (1982) *Drinking and Crime*. New York: Tavistock.

Craig, T. J. (1982) An epidemiologic study of problems associated with violence among psychiatric inpatients. *American Journal of Psychiatry*. 139, 1262–1266.

Eitinger, L. (1978) Psykoser og kriminalitet i Norge. *Nordisk Tidsskrift for Kriminalvidenskab*, 66, 29–47.

Ekblom, B. (1970) Acts of violence by patients in mental hospitals. Dissertation. Uppsala: Scandinavian University Press.

Gottlieb, P., Gabrielsen, G. & Kramp, P. (1987) Psychotic homicides in Copenhagen from 1959 to 1983. *Acta Psychiatrica Scandinavica*, **76**, 285–292.

Heath, D. B. (1983) Alcohol and aggression: a "missing link" in world-wide perspective. In *Alcohol, Drug Abuse and Aggression* (ed. E. Gotthell *et al*, pp. 89–103). Springfield, Illinois, USA: Charles C. Thomas.

Hafner, H. & Böker, W. (1982) *Crimes of Violence by Mentally Abnormal Offenders*. Cambridge: Cambridge University Press.

Krakowski, M., Volavka, J. & Brizer, D. (1986) Psychopathology and violence: a review of the literature. *Comprehensive Psychiatry*, **27**, 131–148.

Kristjansson, E., Allebeck, P. & Wistedt, B. (1987) Validity of the diagnosis schizophrenia in a psychiatric inpatient register. *Nordisk Psykiatrisk Tidsskrift*, **41**, 229–234.

Laoos, J. M., Perlmutter, K. & Saexinger, H. (1977) Fear of the mentally ill: empirical support for the common man's response. *American Journal of Psychiatry*, **134**, 1134–1137.

Miettinen, O. F. (1985) *Theoretical Epidemiology*. New York: John Wiley & Sons.

Mills, M. J. (1988) Civil commitment. The relationship between perceived dangerousness and mental illness. *Archives of General Psychiatry*, **45**, 770–772.

Monahan, J. & Steadman, H. J. (1983) Crime and mental disorder: an epidemiological approach. In *Crime and Justice: An Annual Review of Research*, vol. 4 (eds. M. Tonry & N. Morris), pp. 145–189. Chicago: The University of Chicago Press.

Planansky, K. & Johnston, R. (1977) Homicidal aggression in schizophrenic men. *Acta Psychiatrica Scandinavica*, **55**, 65–73.

Rappeport, J. R. & Lassen, G. (1965) Dangerousness-arrest rate comparisons of discharged patients and the general population. *American Journal of Psychiatry*, **121**, 776–783.

Rossi, A. M., Jacobs, M., Monteleone, M., *et al* (1986) Characteristics of psychiatric patients who engage in assaultive or other fear-inducing behaviors. *Journal of Nervous and Mental Disease*, **174**, 154–160.

Rubin, B. (1972) Prediction of dangerousness in mentally ill criminals. *Archives of General Psychiatry*, **27**, 397–407.

Segal, S. P., Watson, M. A. Goldfinger, S. M., *et al* (1988) Civil commitment in the psychiatric emergency room: The assessment of dangerousness by emergency room clinicians. *Archives of General Psychiatry*, **45**, 748–752.

Steadman, H. J. (1980) The right not to be a false positive: problems in the application of the dangerousness standard. *Psychiatric Quarterly*, **52**, 84–99.

Tanay, E. (1987) Homicidal behavior in schizophrenics. *Journal of Forensic Sciences*, **32**, 1382–1388.

Tardiff, K. & Sweillam, A. (1980) Assault, suicide, and mental illness. *Archives of General Psychiatry*, **37**, 164–169.

Taylor, P. (1982) Schizophrenia and crime. In *Abnormal Offenders, Delinquency, and the Criminal Justice System* (eds. J. Gunn & D. P. Farrington). New York: John Wiley & Sons.

Taylor, P. J. & Gunn, J. (1984) Violence and psychosis. I – Risk of violence among psychotic men. *British Medical Journal*, **288**, 1945–1949.

—— & Parrott, J. M. (1988) Elderly offenders. A study of age-related factors among custodially remanded prisoners. *British Journal of Psychiatry*, **152**, 340–346.

Teplin, L. A. (1985) The criminality of the mentally ill: a dangerous misconception. *American Journal of Psychiatry*, **142**, 593–599.

The Penal Code of Sweden (1972) (original trans. Th. Sellin; amendment trans. J. L. Getz). NJ: South Hackensack.

The Swedish Code of Judicial Procedure, (1968) (trans. A. Bruzelius & R. B. Ginsburg). NJ: South Hackensack.

Virkkunen, M. (1974*a*) Alcohol as a factor precipitating aggression and conflict behaviour leading to homicide. *British Journal of Addiction*, **69**, 149–154.

—— (1974*b*) Observations on violence in schizophrenia. *Acta Psychiatrica Scandinavica*, **50**, 145–151.

Walker, N. (1978) Dangerous people. *International Journal of Law and Psychiatry*, **1**, 37–50.

Weller, M. P. I. & Weller, B. G. A. (1988) Crime and mental illness. *Medicine, Science and the Law*, **28**, 38–46.

Wiersma, D. (1966) Crime and schizophrenics. *Excerpta Criminologica*, **6**, 169–181.

Wikström, P.-O. H. (1985) Everyday violence in contemporary Sweden. *The National Council for Crime Prevention* (report no. 15). Stockholm: Liber Forlag.

Wolfgang, M. E. (1958) *Patterns in Criminal Homicide*. Philadelphia: University of Pennsylvania Press.

World Health Organization (1967) Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death, 1965 revision (8th edn) (ICD-8). Geneva: WHO.

Yearbook of Legal Statistics (1980) *Official Statistics of Sweden*. Stockholm: The National Central Bureau of Statistics.

*Per Lindqvist, MD, *Department of Forensic Psychiatry, University Hospital, Umeå, Sweden, and Department of Forensic Medicine, University of Umeå, Sweden*; Peter Allebeck, MD, PhD, *Department of Medicine, Section for Community Medicine, Karolinska Institute at Huddinge University Hospital, Stockholm, Sweden.*

*Correspondence

British Journal of Psychiatry (1990), 157, 351–354

Langfeldt's Schizophreniform Psychoses Fifty Years Later

A. L. MINA BERGEM, ALV A. DAHL, CATO GULDBERG and HELGE HANSEN

As a result of follow-up studies published in 1937 and 1939, Langfeldt divided schizophrenia into two groups; 'typical schizophrenia' which had a poor outcome, and the 'schizophreniform psychoses' which had a less typical clinical picture of schizophrenia and a good outcome. Langfeldt's cases of schizophreniform psychoses were reclassified according to the ICD-9 and DSM-III-R diagnostic systems. Most of the schizophreniform psychoses did not appear 'schizophrenia-like' at all, but turned out to be mainly affective disorders. Those included in Langfeldt's diagnosis of 'schizophreniform psychoses' were found to be too heterogenous to validate the existence of this syndrome.

Which signs indicate a good outcome in schizophrenia? Ever since the concept was introduced by Bleuler in 1911, psychiatrists have tried to answer that important question. Some have emphasised the symptoms, while others have stressed the duration of the illness or the pre-morbid personality as the most important factor. A recent attempt to delineate a subtype of schizophrenia with good outcome is the introduction of the category 'schizophreniform disorder' in DSM-III (American Psychiatric Association, 1980). This category is based on a combination of typical symptoms and brief duration. Yet, considerable disagreement exists as to whether it is valid (Fogelson *et al*, 1982; Coryell & Tsuang, 1982, 1986; Beiser *et al*, 1988).

Since the present concept seems confusing, a review of its history may provide valuable insight. The term 'schizophreniform', meaning schizophrenia-like, is not new, but was introduced by Langfeldt 50 years ago in 1939. He was the first psychiatrist who tried to separate in a definite way schizophrenias with poor outcome ('typical schizophrenia') from those with good outcome ('schizophreniform psychoses').

In 1926 Langfeldt had suggested that the diagnosis of schizophrenia should be restricted to the group of disorders which, as proved by individual follow-up examinations, had a poor outcome. The other group he had termed as 'doubtful schizophrenia'. These two groups had also been separated at the University Psychiatric Clinic in Oslo from its opening in 1926: a diagnosis of 'typical schizophrenia' there meant belonging to the central dementia praecox group with poor outcome. If there was any doubt, one or more question marks were added to the diagnosis category (i.e. the term 'schizophrenia (?)' was employed). When Langfeldt attended the Clinic in 1935, he decided to compare the natural course of these two types of schizophrenia. Thus, he conducted personal follow-up examinations of 200 cases whose case records showed a diagnosis, given between 1926 and 1929 by the Clinic, of 'typical schizophrenia' (100 cases) or 'schizophrenia (?)' (100 cases) (Table I). He found, however, that some of the 'typical' cases diagnosed by the Clinic, when reclassified according to the follow-up data, were atypical (Langfeldt, 1937). He termed these atypical cases 'Schizophreniform psychoses' and since they showed symptoms and personality traits that were reminiscent of true schizophrenia, even though they lacked the central, primary symptoms typical of the classical dementia praecox. He also found that the 'schizophrenia (?)' group had a much better outcome than the typical schizophrenia one (see Table I).

Langfeldt stated clearly that the 'schizophreniform psychoses' were a heterogeneous diagnostic category with only two things in common: *the symptom picture was close to that of schizophrenia, and the outcome was good.* On that basis he described five different subtypes of such psychoses: (1) preponderantly endogenically conditioned (constitutional) forms, (2) neurosis-like, psychogenically more comprehensible cases, (3) symptomatic and other predominantly exogenically (infectiogenic, traumatogenic, psychogenic) precipitated psychoses, (4) cases belonging to the manic–depressive group of psychoses, and (5) real atypical schizophrenias (Langfeldt, 1982).

Langfeldt's description of the schizophreniform group is cast in outdated diagnostic terms. We therefore decided to reinvestigate the original 100 'schizophrenia (?)' cases by means of two modern classification systems, ICD-9 (World Health Organization, 1978) and DSM-III-R (American Psychiatric Association, 1987), and thereby examine the validity of his concept of 'schizophreniform psychoses'.

TABLE I

Outcome of 100 cases of 'typical schizophrenia' and 100 cases of 'schizophrenia(?)', originally diagnosed by the University Psychiatric Clinic, Oslo, and followed-up and reclassified by Langfeldt

Psychiatric Clinic diagnoses (1926–29)	*Langfeldt's diagnoses (1936)*	*Outcome*			
		Unchanged/ worse	*Improved*	*Recovered*	*Total*
Typical schizophrenia ($n = 100$)	Schizophrenia	66	15	6	87
	Schizophreniform	0	2	11	13
Schizophrenia (?) ($n = 100$)	Schizophrenia	36	8	1	45
	Schizophreniform	7	17	31	55

Method

Four psychiatrists worked as a team in reclassifying the 100 cases with an original diagnosis of 'schizophrenia (?)'. The records of each case were examined, and the psychopathological data on these turned out to be of high quality. Since it was considered too time-consuming for each rater to read all the records the case summary method was used in which each member of the team prepared 25 case summaries of one-and-a-half to three pages long. Background and life-history of each case were stated briefly, and then the descriptions of the development of the illness and the psychopathology were quoted in full. When basic symptoms of psychoses were not mentioned in the records, we counted them as non-existent. In 90 of the 100 cases the records also contained follow-up information, but this was noted separately from the case summaries. At this point only the information known at the index admission to hospital was used as the basis for diagnosis – this was what was available to Langfeldt before he followed up the cases.

Using the case summaries, diagnoses were made by each of the team members according to DSM–III–R and ICD–9. Only one principal diagnosis in each system was allowed in each case. The team had regular meetings in which their individual diagnoses in the two systems were presented and a consensus diagnosis on each case was reached. The kappa coefficients between pairs of raters and between raters and consensus were calculated to check inter-rater reliability. The proportion of complete agreement to no agreement among the raters was also calculated (Table II).

Results

For the calculation of inter-rater reliability, diagnoses on the three-digit level in both the DSM-III-R and ICD-9 classification systems were used. The inter-rater reliability of the diagnoses based on the case summaries were close to 0.50 in both systems (see Table II). The agreement between the raters and consensus were close to 0.70. All four raters were in full agreement on the diagnosis of 41 and 42 cases in DSM–III–R and ICD–9 respectively. In DSM–III–R three raters agreed on the diagnosis of 42 patients, while the corresponding number was somewhat lower in ICD–9.

TABLE II

Agreement on the diagnoses by the psychiatric team members

	DSM–III–R	*ICD–9*
	kappa	*kappa*
Inter-rater agreement range	0.47–0.58	0.47–0.55
Rater-consensus agreement range	0.63–0.76	0.64–0.74
Inter-rater agreement range on schizophrenia v. affective disorders	0.72–0.88	0.67–0.85
	n	*n*
All four raters agree on diagnosis	41	42
Three raters agree on diagnosis	42	33
Two raters agree on diagnosis	17	24
All raters disagree on diagnosis	0	1

When diagnoses were made on the three-digits level, 10 disorders were found in both classification systems. A considerable heterogeneity of diagnoses were observed when the 'schizophreniform psychoses' were classified according to DSM–III–R and ICD–9 (Tables III and IV). This is in accordance with Langfeldt's conceptualisation.

Langfeldt classified 45 of the 'schizophrenia (?)' patients as true schizophrenics on follow-up examination (Table I). We found the proportion of true schizophrenia somewhat lower in this 'schizophrenia (?)' group, but we also found that some cases with other index diagnoses developed a more typical picture of schizophrenia during the follow-up period.

Langfeldt's first premise for 'schizophreniform psychoses', namely that the clinical picture should be schizophrenia-like, does not seem to be true for the 55 cases he classified in this category. Most of these cases have various affective disorders. Their clinical picture does not resemble schizophrenia, although cases with mood-incongruent psychotic features can sometimes be mistaken as having that disorder. However, only twelve of the psychotic cases with bipolar or major depressive disorder had those features. The

TABLE III

DSM-III-R consensus diagnoses of Langfeldt's 100 cases of 'schizophrenia (?)'

Diagnosis	n
Alcoholism	2
Schizophrenia	19
Schizophreniform disorder	14
Major depression	33
Bipolar disorder	8
Delusional disorder	3
Brief reactive psychosis	2
Psychotic disorder not otherwise specified	4
Conversion disorder	1
Social phobia	1
Hypochondriasis/somatoform disorder	2
Schizotypal personality disorder	2
Personality disorder not otherwise specified	2
Adjustment disorder	2
Depressive disorder not otherwise specified	5
Total	100

TABLE IV

ICD-9 consensus diagnoses of Langfeldt's 100 cases of 'schizophrenia (?)'

Diagnosis	n
Alcohol psychosis	1
Transient organic psychosis	1
Schizophrenia, acute	14
Schizophrenia, subchronic/chronic	18
Schizophrenia, schizoaffective	4
Manic-depressive psychosis, manic	2
Manic-depressive psychosis, depressed	28
Manic-depressive psychosis, circular	5
Paranoid psychoses	3
Reactive psychoses	9
Psychosis *non ultima descriptum*	2
Hysteria	1
Phobic state	1
Neurotic depression	3
Hypochondriasis	2
Schizoid personality disorder	2
Personality disorder *non ultima descriptum*	2
Adjustment disorder	2
Total	100

schizoaffective category of ICD-9 (295.7) gives an opening for classifying such intermediate cases, but the number of cases given this diagnosis was also quite low (Table IV).

Langfeldt's second premise, that of good outcome, we presume to hold true. The proportion of the 'schizophrenia (?)' cases ($n=55$) which Langfeldt called 'schizophreniform psychoses' was made up by those with affective disorders, reactive psychoses, neuroses and adjustment disorders. These disorders often show a good outcome.

Cases with either schizophrenia (295) or major affective disorder (296) in the modern classifications constitute the major part of Langfeldt's sample ($n>70$). The kappa coefficients for the raters on the classification of schizophrenia v. affective disorder are in the range of 0.67 to 0.85 (Table II), which indicate a higher inter-rater agreement. Discussions of disagreements before consensus diagnoses were made add further support to the correct classification of these cases.

Discussion

The case-summary method was chosen for this study. In this, all the raters have the same diagnostic information, but still the inter-rater agreement between pairs of raters was only moderate. The main reason for this seems to be that the raters interpreted the recorded psychopathology in different ways since old-fashioned terms sometimes were used. However, consensus after discussion corrects for such errors. Furthermore, minimal differences in the diagnostic agreements were found when the DSM-III-R and ICD-9 systems were compared. In addition, the basic separation of schizophrenia and major affective disorder, which comprise about three-quarters of the cases, shows a high inter-rater agreement, and at least three of the raters were in agreement concerning the three-digit diagnoses in the same high proportion (75%) of cases. Taken together, these factors support the validity of our classifications.

Like Langfeldt we did not see the patients at their index admission to hospital. However, he saw them at follow-up, and decided that 45 of them had 'typical' schizophrenia. From the follow-up information, we know that most of the cases which we diagnosed as having acute schizophrenia (ICD-9) or schizophreniform disorder (DSM-III-R) turned out to become chronic cases with poor outcome. Our diagnoses of subchronic and chronic schizophrenia therefore seem close to Langfeldt's concept of 'typical' schizophrenia. It is reasonable to assume that a few of the other cases also developed schizophrenia during the follow-up period or were misclassified in the first place.

Among the 55 cases called 'schizophreniform' by Langfeldt at follow-up, most seem to have an affective disorder. Others have a neurosis or an adjustment disorder. These disorders are known to have a good outcome, a fact also confirmed by Langfeldt. However, the clinical picture of these cases is definitely not 'schizophrenia-like' as suggested by him. His basic premise for the term 'schizophreniform' does not hold true. The good outcome of Langfeldt's 'schizophreniform psychoses' is better explained by the fact that most of them are affective disorders. One could argue that the concept of affective disorders has expanded or changed over the years, thereby making Langfeldt right anyway. However, Norwegian psychiatrists at that time knew Kraepelin's works very well, and the Norwegian concept of schizophrenia has always been a narrow one.

We found just 12 cases of affective disorder with mood-incongruent psychotic features in DSM-III-R, which probably could be seen as schizophrenia-like.

Conclusions

Fifty years have passed since Langfeldt published his influential studies on schizophrenia. His point of departure and main message sounds refreshingly up to date: "If researchers on schizophrenia, in reporting their findings, had always indicated the special type and mental state of the disorder instead of merely using the term 'schizophrenia', the progress in research on these sorts of disorders would probably have been much greater." (Langfeldt, 1939). He contributed to this aim himself by introducing the dichotomy of 'typical schizophrenia' with poor outcome and 'schizophreniform psychoses' with good outcome. His criteria for 'poor outcome' schizophrenia are still unsurpassed fifty years later (Brockington *et al*, 1982). The basis for his concept of 'schizophreniform psychoses' as good-outcome schizophrenia does not hold up, however. According to our reclassification, most of these cases do not look like schizophrenia. The term 'schizophreniform' therefore seems unjustified.

Langfeldt investigated a basically untreated patient sample. Since it is impossible to find such a sample today, the study of such old ones may help provide answers to the question which, like Langfeldt 50 years ago, we are still asking today: that is, what *are* the criteria of 'good-outcome' schizophrenia?

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorder* (3rd edn) (DSM-III). Washington, DC: APA.

—— (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

Beiser, M., Fleming, J. A. E. & Lin, T. (1988) Refining the diagnosis of schizophreniform disorder. *American Journal of Psychiatry*, **145**, 695–700.

Brockington, I. F., Kendell, R. E. & Leff, J. P. (1982) Definitions of schizophrenia: concordance and prediction of outcome. *Psychological Medicine*, **8**, 387–398.

Coryell, W. & Tsuang, M. T. (1982) DSM-III schizophreniform disorder. *Archives of General Psychiatry*, **39**, 66–69.

—— & —— (1986) Outcome after 40 years in DSM-III schizophreniform disorder. *Archives of General Psychiatry*, **43**, 324–328.

Fogelson, D. L., Cohen, B. M. & Pope, H. G. (1982) A study of DSM-III schizophreniform disorder. *American Journal of Psychiatry*, **139**, 1281–1285.

Langfeldt, G. (1926) *The Endocrine Glands and the Autonomic Systems in Dementia Praecox*. Bergen: Eide.

—— (1937) *The Prognosis in Schizophrenia and the Factors Influencing the Course of the Disease*. London: Melford.

—— (1939) *The Schizophreniform States*. Copenhagen: Munksgaard.

—— (1982) Definition of "schizophreniform psychoses". *American Journal of Psychiatry*, **139**, 703.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

A. L. Mina Bergem, MD, *Research Psychiatrist, Department of Psychiatry, University of Oslo, PO Box 85, Vindern, 0319 Oslo 3, Norway*; Alv A. Dahl, MD, *Associate Professor, Department of Psychiatry, University of Oslo*; Cato Guldberg, MD, *Senior Registrar, Department of Psychiatry, University of Oslo*; Helge Hansen, MD, *Registrar, Department of Psychiatry, University of Oslo*

British Journal of Psychiatry (1990), **157**, 355–358

Impaired Ability of Schizophrenics, Relative to Manics or Depressives, to Appreciate Social Knowledge about their Culture

J. CUTTING and D. MURPHY

Twenty schizophrenics, 20 manics, and 20 depressives were given two sets of multiple choice questions, one testing the subject's social knowledge of how people tend to act in a social situation and the other tapping their knowledge of events or objects which are relatively free of a social component. Schizophrenics were significantly impaired on the former set of questions relative to manics, and were significantly worse on both than depressives. It is suggested that these results represent an objective measure of the social naïvete of schizophrenics. The significant difference from manics indicates that the results are not merely the general effects of psychosis, particularly because the manics performed worse on an attentional test than the schizophrenics.

In a previous article (Cutting & Murphy, 1988), we described the development of a multiple choice questionnaire to measure one aspect of schizophrenic thought disorder. This comprised 36 questions: 12 from the 'cognitive estimates test' of Shallice & Evans (1978), purportedly sensitive to frontal lobe damage; 12 questions which we devised and standardised ourselves, aimed at tapping a subject's general knowledge of the state of the world – 'everyday knowledge'; and 12 personally devised and standardised questions on a subject's knowledge of the relative likelihood of alternative behaviours of people in specific social situations – 'social knowledge'. Examples of these are provided in the Appendix.

In the previous article we presented the results of 20 schizophrenics and 30 neurotic controls on the last two sets of questions. The schizophrenics were markedly inferior to the controls on both sets of questions, and we therefore suggested that the combined sets of questions might provide a useful measure of schizophrenic thought disorder. They certainly discriminated between the two groups better than conventional tests of thought disorder such as incorrect proverb interpretation, overinclusiveness, illogical reasoning on syllogisms, or conceptual loosening.

However, we were limited in our conclusions by the nature of the control group. In this study we attempted to remedy this by administering the questions to subjects suffering from a depressive or manic illness. The criticism of most previous purported measures of schizophrenic thought disorder is that they fail to discriminate manic from schizophrenic thought disorder.

Method

Subjects

Twenty schizophrenics, 20 manics and 20 subjects with major depressive disorder, all diagnosed according to the Research Diagnostic Criteria (RDC) of Spitzer *et al* (1975), were tested. The subjects were all recently admitted in-patients to the Bethlem and Maudsley Hospitals. The schizophrenics were, as closely as could be achieved, consecutive admissions with this diagnosis under the care of the senior author (although a handful were missed because of holidays of DM who tested them). The depressives were, with the above proviso, consecutive admissions under the care of another consultant specialising in the management of depression. The manics were consecutive admissions with this diagnosis in the intensive care ward of the hospital. Schizoaffectives were excluded because of the nosological uncertainty of this diagnosis. The schizophrenics had either acute ($n = 12$) or subacute ($n = 8$) illnesses according to RDC criteria, with a mean of 2.2 discrete psychotic episodes. The schizophrenics were significantly younger (27.9 years) than the manics (37.8 years) or depressives (41.5 years). The sex distribution of the groups (schizophrenics – 13 men, 7 women; manics – 11 men, 9 women; depressives – 10 men, 10 women) was, however, comparable. An estimate of pre-morbid intelligence (Nelson & O'Connell, 1978) also revealed no significant differences between the mean intelligent quotients (IQs) for the three groups – schizophrenics 113.5, manics 112.6, depressives 109.4. The IQs of all three groups, although comparable, are higher than average. There are two explanations for this. Firstly, Nelson & O'Connell's IQ equivalents were based on the old Wechsler Adult Intelligence Scale (WAIS) and the new revised WAIS (WAIS-R) has shown that the old WAIS overestimated the average IQ by six points. Secondly, patients are often admitted to the Bethlem and the Maudsley Hospitals, particularly to the specialist units, as a result of a referral for a second opinion. They often therefore come from

TABLE I
Schizophrenics', manics' and depressives' mean scores (s.d.) on multiple choice questionnaire (MCQ) and attentional tasks

	Schizophrenics (n = 20)	*Manics* (n = 20)	*Depressives* (n = 20)
Total MCQ score/18	11.5 (3.4)	13.2 (3.4)	16.4*** (1.5)
subset social questions/9	5.1 (2.3)	6.8* (1.8)	8.4*** (0.8)
subset non-social questions/9	6.4 (1.7)	6.4 (1.9)	8.0** (1.0)
Attentional tasks			
errors on 100–7 test/5	1.9 (1.9)	1.6 (1.7)	1.2 (0.9)
errors on digit cancelling/21	2.1 (2.1)	4.8** (6.9)	0.9** (0.7)

*$P<0.05$, **$P<0.01$, ***$P<0.001$ (*t*-test between schizophrenics and another group).

middle-class families who have had the knowledge and determination to institute the referral. Thus, they are likely to have a higher IQ than average.

Procedure

In our previous study we administered 36 questions to 20 schizophrenics and 30 neurotic patients. For the present study, we retained 18 of these which best discriminated between the two groups – those where the mean 'correct' neurotics' answers minus mean 'correct' schizophrenics' answers was greatest ('correct' in this context means that the answers supplied were those which the normal standardisers had given – see Cutting & Murphy, 1988). Included in these 18 were four of the 'cognitive estimate' questions, six of the original 'everyday knowledge' questions and eight of the 'social knowledge' questions. On reconsideration, one of the 'everyday knowledge' questions ('If you won the Football Pools . . .', see Appendix) appeared to be more appropriate in the social category, and so the 18 questions divided neatly into nine with a minimal and nine with a substantial social component. Although one can quibble about the extent of the social component in some of them, the distinction seemed important because we previously found (Cutting & Murphy, 1988) that it was precisely on those questions which examined a subject's knowledge of how other people might behave in a certain situation that schizophrenics deviated most from the norm. We wished to confirm or refute this in the present study.

To guard against the possibility that deviant performance by the schizophrenics might be merely an effect of inattention, we also administered two tests of attention: the 100–7 test scored according to the recommendations of Folstein *et al* (1975) in their Mini-Mental State Examination; and crossing out 21 digit 3s in a matrix of 169 random digits, a test generally considered to be a valid measure of attention.

The 18-item multiple choice questionnaire and the attention test were administered in one sitting. No practice trials were given for either. In the case of the questionnaire, subjects were told that they would be asked some questions about how they might react in certain circumstances and then shown four alternative solutions. It was stressed to them that there was no right or wrong answer – it was their opinion which the experimenter wanted. No feedback was given whether they were 'correct' or 'incorrect'.

Results

The results (Table I) demonstrate that the schizophrenics were significantly inferior to the depressives on the entire set of 18 questions and on each subset. However, relative to the manics, the schizophrenics were only significantly impaired on the set of questions where we had attempted to maximise the social component. This difference between schizophrenics and manics could not be attributed to the former group being less attentive, because the manics made significantly more errors on the digit cancelling test.

Discussion

The aim of this study was to refine and validate a measurement of schizophrenic thought disorder which had looked promising in an earlier study. We were partially successful in achieving this aim, in that a subset of questions did allow a significant discrimination between schizophrenics and manics. Although the difference was only significant at the 5% level, given that there were only nine questions involved, this was a considerable achievement. The sensitivity of the test at a score of five or less 'correct' answers out of a total of nine was 55%, and its specificity with respect to excluding manics at this cut-off was 80%. We cannot, therefore, recommend the test as it stands as a powerful diagnostic tool, but the fact that so few questions produced any significant difference at all between two groups notoriously difficult to distinguish psychometrically is promising. The potential value of the study, in our view, is the fact that a particular thematic content was responsible for the relatively deviant schizophrenic answers. It is possible, therefore, that a larger number of questions on the same theme might increase the sensitivity and specificity of the test.

If the test is tapping a particular lacuna in a schizophrenic's world view, what is the nature of this?

One possibility is that schizophrenics fail to make appropriate judgements about the affairs and behaviour of others through lack of exposure to sufficient examples, or through social isolation (self-imposed or enforced by frequent admission to hospital). In other words, their social naïvete is a consequence of environmental factors, interconnected with their personality or actual illness, but not a core feature of their illness. This probably broadly represents the views of Wing & Brown (1970), Ciompi (1983), and other social psychiatrists.

An alternative view, which we espouse, is that incompetent social judgements of this type assessed here are an intrinsic feature of schizophrenia. Minkowski (1927) and Cameron (1944) were prominent exponents of this approach.

The present study was not specifically designed to clarify which of these alternative views is more correct. However, by subdividing the schizophrenics in our study in various ways it is potentially feasible to examine this issue. For example, if frequent admission to hospital played a substantial role in a subject's impoverished social knowledge, then a group with more than one admission should have a lower social knowledge score than a group tested in their first admission. In fact, this is so. The eight schizophrenics in their first admission had a mean score on the social questions of 5.5, compared with a mean score of 4.8 by the 12 who had previous hospital admissions. This was not significant, however, and its implications are undermined by two further factors. Firstly, the 12 schizophrenics with an acute onset to their current episode had a mean score of 4.8 on the social questions, whereas the eight with a subacute onset had a corresponding mean score of 5.5. Secondly, dividing subjects up into paranoid and hebephrenic subgroups according to the criteria of Tsuang & Winokur (1974), the latter subgroup had a lower score than the former one (5.5). Overall, therefore, it is not certain that prolonged social isolation contributed to the impoverishment in social knowledge, because acuteness of psychosis and the presence of hebephrenic signs were contributory factors. It is possible that the deficient social knowledge is restricted to a subgroup of schizophrenics, in which case it would not be legitimate to speak of a distinctively schizophrenic world view.

Even if the poor performance could be shown to be an intrinsic feature of schizophrenia, or of an identifiable subgroup with this condition, it may still not be justifiable to refer to this as 'incompetent social judgements'. It might be that such schizophrenics are not generally inept in such matters, but follow certain rigid rules which may or may not be appropriate in the circumstances. They might be over-influenced by some detail in the scene in front of them, if Shakow's (1950) views on the psychological deficit in schizophrenia are correct. This might make their judgements erratic and sometimes inappropriate. Alternatively, they may behave according to the immediacy hypothesis of Salzinger *et al* (1970), overly determined by the first cue they receive, with little heed to the overall context of the scene.

The purpose of this study was rather to record than to explain certain behaviour in schizophrenia, and relevant hypotheses could be examined in subsequent studies. We hope that the results reported here, particularly the significant difference between schizophrenics and manics, will stimulate further research.

Appendix

Social knowledge questionnaire

(a) Why do you think the divorce rate is going up?
  (i) Rising birth rate.
  (ii) Too many police around.
  (iii) Rise in the cost of living.
  (iv) Divorce laws are less strict.*
(b) What do you think would be the main consequence if suddenly there were no police?
  (i) More suicides.
  (ii) More people on the streets.
  (iii) Fewer cops and robbers films on the TV.
  (iv) More burglaries.*
(c) What do you think would be the most sensible thing to say if you came across two strangers having a fight in the street?
  (i) I suppose you think you're clever.
  (ii) Stop it. You'll lose your jobs.
  (iii) Stop it. You'll upset your mothers.
  (iv) The police are just around the corner.*
(d) Why do you think that some men gamble excessively?
  (i) They need continual excitement.*
  (ii) They had overprotective mothers.
  (iii) They enjoy losing money.
  (iv) They are of low intelligence.
(e) How would you tell a friend politely that they had stayed too long?
  (i) You'd better go. I'm fed up with you staying too long.
  (ii) Haven't you got anything better to do.
  (iii) Excuse me. I've got an appointment with a friend.*
  (iv) There's no more coffee left.
(f) What helpful thing could you say to a friend who was continually being harassed by their neighbour?
  (i) Don't worry. I've heard she is unpleasant to everybody.*
  (ii) Why don't you throw some rubbish over the wall.
  (iii) You probably deserve it. You're a noisy person.
  (iv) There's nothing you can do. You'll have to move.

(g) Why do you think identical twins are emotionally close?
  (i) They're brought up on the same food.
  (ii) They share the same genetic make-up.*
  (iii) They probably get the same presents at Christmas.
  (iv) No-one will play with them because they're so strange.

(h) What sort of people do you think make the best solicitors?
  (i) People who are angry with the way the country is run.
  (ii) People who like talking a lot.
  (iii) People who don't drink or smoke.
  (iv) People with an eye for detail.*

(i) If you won the Football Pools how best would you guarantee a large income for the rest of your life?
  (i) Buy shares in an Australian gold-mine.
  (ii) Put it on the favourite in next year's Derby.
  (iii) Give it to your neighbour to look after.
  (iv) Place it with an investment company in the City.*

Examples of non-social questions

(from cognitive estimates)
What is the age of the oldest person in Britain today? (normative range 102–112)

(from 'everyday questions')
Why is it unsafe to drink tap water in some countries?
  (i) It's against the law.
  (ii) The water's a murky colour.
  (iii) It might contain typhoid germs.*
  (iv) There might be small insects in it.

References

Cameron, N. (1944) Experimental analysis of schizophrenic thinking In *Language and Thought in Schizophrenia* (ed. J. S. Kasanin), pp. 50–64. Boston: Houghton Mifflin & Co.

Ciompi, L. (1983) Schizophrenic deterioration. *British Journal of Psychiatry*, **143**, 79–80.

Cutting, J. & Murphy, D. (1988) Schizophrenic thought disorder. *British Journal of Psychiatry*, **152**, 310–319.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) Mini-mental scale. A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198.

Minkowski, E. (1927) The essential disorder underlying schizophrenia and schizophrenic thought (trans. 1987). In *The Clinical Roots of the Schizophrenia Concept* (1987) (eds J. Cutting & M. Shepherd), pp. 188–212. Cambridge: Cambridge University Press.

Nelson, H. E. & O'Connell, A. (1978) Dementia: the estimation of premorbid intelligence levels using the new adult reading text. *Cortex*, **14**, 234–244.

Salzinger, K., Portnoy, S., Pisoni, D. B., *et al* (1970) The immediacy hypothesis and response-produced stimuli in schizophrenic speech. *Journal of Abnormal Psychology*, **76**, 258–264.

Shakow, D. (1950) Some psychological features of schizophrenia. In *Feelings and Emotions* (ed. M. L. Reymert), pp. 383–390. New York: McGraw-Hill.

Shallice, T. & Evans, M. E. (1978) The involvement of the frontal lobes in cognitive estimation. *Cortex*, **14**, 294–303.

Spitzer, R. L., Endicott, J. & Robins, E. (1975) *Research Diagnostic Criteria*. New York: New York State Psychiatric Institute.

Tsuang, M. T. & Winokur, G. (1974) Criteria for subtyping schizophrenia. *Archives of General Psychiatry*, **31**, 43–47.

Wing, J. K. & Brown, D. W. (1970) *Institutionalism and Schizophrenia*. Cambridge: Cambridge University Press.

*J. Cutting, MD, MRCPsych, MPhil, MRCP, *Consultant Psychiatrist, Bethlem Royal Hospital, Monks Orchard Road, Beckenham, Kent*; D. Murphy, MB, MRCPsych, *Senior Registrar, Bethlem Royal Hospital*

*Correspondence

British Journal of Psychiatry (1990), **157**, 359–365

Regional Cerebral Blood Flow in Patients with Affective Disorders

V. DELVENNE, F. DELECLUSE, Ph. P. HUBAIN, A. SCHOUTENS, V. De MAERTELAER and J. MENDLEWICZ

Regional cerebral blood flow at rest was measured in 38 patients with major depressive disorders and 16 controls by SPECT with inhalation of xenon-133. All subjects had been withdrawn from medication. The mean hemispheric cerebral blood flow was not statistically different between the controls and the different subgroups of depressed patients defined either by biological markers or clinical characteristics. However, the predominantly cortical blood flow, measured on the outer cerebral rim of the third tomographic slice, was significantly lower on the left hemisphere in bipolar patients when compared with normals and unipolar patients. The same lateralisation was observed in patients with an endogenous depression according to the Newcastle scale.

Cerebral localisation and hemispheric lateralisation of mental dysfunction have been the subject of several investigations (D'Elia & Perris, 1973; Flor-Henry, 1976; Gruzelier & Flor-Henry, 1979). Studies of mood changes following brain lesions suggest an asymmetrical functional regulation of emotional behaviour: damage to the left hemisphere seems to be associated with depression, whereas lesions on the right are associated with euphoria or indifference (Lishman, 1968; Sackeim *et al*, 1982; Lipsey *et al*, 1983).

Regional cerebral blood flow (rCBF) can be estimated by scintigraphy with inhaled xenon-133, a non-invasive, reliable and reproducible technique (Obrist *et al*, 1975). Several studies have used this method to try to demonstrate cerebral dysfunction in depressed (and other) patients, but different methodology and patient populations have led to conflicting results: a decrease in rCBF at rest (Mathew *et al*, 1980; Gustafson *et al*, 1981); a decrease in rCBF after electroconvulsive therapy (ECT) (Johanson *et al*, 1979; Silfverskiöld *et al* 1979); a hypervascularisation of the left frontal lobe (Uytenhoef *et al*, 1983); and negative results (Risberg, 1980; Gur *et al*, 1982, 1984; Silfverskiöld & Risberg, 1989). In the present study, we used a tomographic xenon-133 inhalation technique to measure rCBF in unmedicated depressive patients and normal controls.

Method

All consecutive patients admitted during 1987 to the psychiatric department of Erasme Hospital for treatment of a depressive illness were considered for this study. Those studied (17 men and 21 women) were free from alcoholism, drug abuse, and concomitant neurological or medical diseases, and had not been treated with ECT, monoamine oxidase inhibitors, or lithium carbonate for at least two months before the study. All subjects were submitted to a drug wash-out of at least 10 days for benzodiazepines and antidepressants, and at least 20 days for neuroleptics.

All patients had a major depressive disorder according to Research Diagnostic Criteria (RDC; Spitzer *et al*, 1975). With the same criteria, they were classified into various clinical subgroups: primary versus secondary depressive disorders, endogenous versus non-endogenous, unipolar versus bipolar type. The severity of depression was quantified by the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) on the day of rCBF measurement. The score had to be 15 or greater for patients to be included in the study. The Newcastle scale (Carney *et al*, 1965) was used to examine endogenicity.

Biological markers of depression were also examined. Sleep electroencephalography (EEG) was performed two or three nights after one or two accommodation nights. They were visually scored according to standard criteria (Rechtschaffen & Kales, 1968; Mendlewicz *et al*, 1984) by an experienced assessor, rapid eye movement (REM) latency being defined as the time between sleep onset and first REM stage. The dexamethasone suppression test (DST) was performed two days before EEG, as it has been shown that dexamethasone administration within this time does not affect sleep (Kupfer *et al*, 1978). Dexamethasone (1 mg) was given orally at 11.00 p.m. and blood was drawn the next day at 4.00 p.m. and 11.00 p.m. for cortisol assay. Suppression was defined on the basis of a plasma cortisol level at 4.00 p.m. and/or 11.00 p.m. under 50 μg/l (Caroll *et al*, 1976).

The controls (eight men, eight women) underwent an identical screening procedure except for sleep EEG and DST; they were also screened for personal and family history of psychiatric disorder. A standard questionnaire was used for patients and controls to determine handedness (Oldfield, 1971).

All patients and controls were requested to give informed consent before inclusion in the study.

Evaluation of cerebral blood flow

Cerebral blood flow was studied by single-photon emission computerised tomography (SPECT) with inhalation of xenon-133. This method has been described in detail by Lassen & Rommer (1986), and only a brief description is given here. The tomograph (Tomomatic 564 spect) consists of four arrays of 64 scintillation detector crystals rotating every 10 seconds around a patient's head. A complete CBF study takes 10 minutes, 120 mCi of ^{133}Xe in a 4l mixture of air and oxygen being inhaled in a closed circuit for the first 1.5 minutes. Five transaxial brain slices were studied simultaneously, each by a sequence of four consecutive one-minute scans. An arterial cerebral input curve was estimated by a lung curve obtained by placing a narrowly collimated detector over the apex of the right lung. All studies were done in a dark, quiet room, with the patient's eyes closed but ears not occluded. End-tidal $p\mathrm{CO}_2$ was measured after a forced expiration during the fifth minute of the examination by means of an infrared capnograph. Head placement was determined by a positioning laser, in reference to the orbitomeatal line (OM). An algorithm based on a combination of the sequential tomographic method (Celsis *et al*, 1981) and the early-picture method (Kanno & Lassen, 1979), followed by correction for tissue absorption of the radiation, allowed the calculation of CBF.

Two parameters (a) and (b) were derived from the flow maps:

(a) *Mean right and left hemispheric flow values*

The outer limit of brain was delineated as the 40% of maximum activity isocontour (Fig. 1). Mean left and right hemispheric CBF were calculated in a standard manner within this outline for the third tomographic slice (slice 3, at OM + 5 cm, which includes basal ganglia, frontal and occipital poles). If airway contamination by xenon was visible on this slice, the following slice (slice 4, at OM + 7 cm) was used instead, in order to avoid artificially increased frontal flow values. We thus obtained left and right rCBF (ml/100 g brain tissue/min). These absolute flow values were corrected for $p\mathrm{CO}_2$ deviation from 40 mmHg, according to the formula proposed by Alexander *et al* (1964) and validated by Rootwelt *et al* (1986): $\mathrm{CBF}_{40} = \mathrm{CBF}_{\mathrm{actual}}\ (1.37 \times 40 - 5.08)/1.37 \times p\mathrm{CO}_2 - 5.08)$.

(b) *Right and left cortical flow values*

The internal delineation of the cerebral rim on slice 3 was manually adjusted to include the high blood flows of the occipital and frontal poles (Fig. 1). This rim thus predominantly, but not exclusively, consisted of cortical areas of the brain. Resolution of the image was 64 × 64 pixels. Blood flow per pixel was calculated for 64 angular positions (individual angle: 2.81°) from frontal to occipital pole along the right and left half rims (Fig. 2). This mode of quantification is based on a method developed in nuclear cardiology (Berman *et al*, 1981). Left–right differences were calculated for each angle and two parameters were derived (Fig. 3):

(i) the sum of left–right differences for the whole rim
(ii) the number and the localisation of peak differences exceeding 10 ml/min/100 g/pixel.

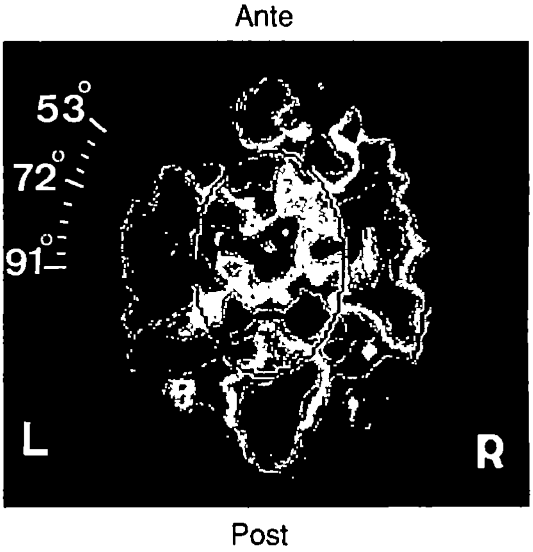


FIG. 1 Cortical blood-flow map from slice 3 illustrating the left–right asymmetry in a bipolar patient. The zone marked (53°–91°) indicates the usual localisation of the peak differences (mean, s.d. = 72°, 19°).

Statistics

Left and right CBF, CBF in the outer rim of slice 3 (all corrected for $p\mathrm{CO}_2$), left–right asymmetry, and localisation of this asymmetry were compared between the normal control group, the whole patient population, and the different clinical or biological patient subgroups. The same comparisons were performed for right-handed subjects only.

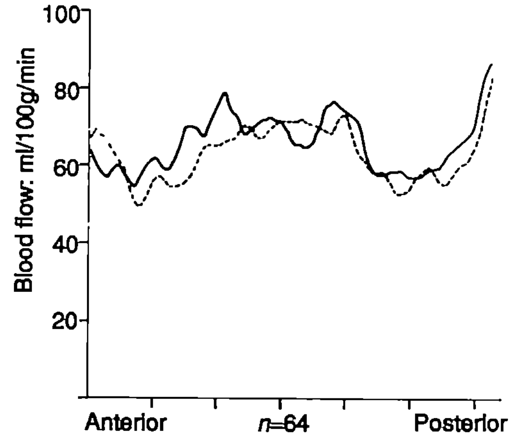


FIG. 2 Flow profiles in right (——) and left (– – –) halves of the outer rim of the horizontal slice 3 in the same patient as in Fig. 1.

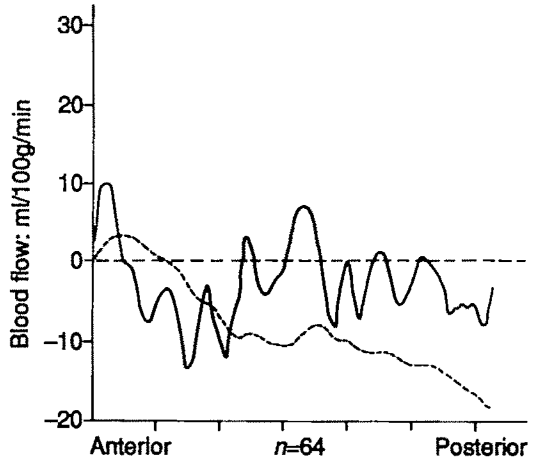


Fig 3 Left–right differences in flow (——) and the integral of these differences for the all rim (– – –) in the same patient as in Figs 1 and 2.

The continuous variables were analysed by comparing group means with a one-factor analysis of variance (when three groups were involved) followed when necessary with contrast analysis or with a Student's *t*-test. Categorical variables (number of peak differences) were analysed with χ^2 or Fisher tests. Computations were performed with the use of the SPSS statistical software (Nie *et al*, 1975).

Results

The demographic and clinical characteristics of the 38 patients meeting the entry criteria and the 16 controls are presented in Table I. Controls and depressive patients did not differ in mean age or sex distribution. None of the variables investigated (left rCBF, right rCBF, total rCBF, rCBF of the outer rim) were found to be related to either age, sex or Hamilton score. End-tidal pCO_2 did not differ in the different groups. Table I also shows the means and standard deviations of left, right and total hemispheric CBF in each subgroup of depressed patients and in controls. No statistical differences were observed between groups.

Cortical cerebral blood flow measured in the outer rim of slice 3 is presented in Table II. There was significant asymmetry (left < right) in bipolar patients compared with unipolars ($P = 0.019$) and with controls ($P = 0.038$). The same lateralisation was observed in endogenous patients according to the Newcastle scale, who differed significantly from non-endogenous patients ($P = 0.026$) and less significantly from controls ($P = 0.074$). These differences were confirmed when only right-handed people were considered. Table II also presents the number of peaks (left–right differences) in slice 3. No significant difference in peak number or localisation was observed between the normal subjects and the different clinical subgroups of patients. The peak differences, for all the groups (patients and controls), was situated at 72° ± 19° (mean ± s.d.).

Figures 1–3 illustrate the third tomographic slice and the left–right asymmetry of a bipolar patient.

Discussion

Most previous studies reported controversial results of rCBF in depression. Johanson *et al* (1979), using the xenon-inhalation technique with 16 collimated probes and with pCO_2 correction, examined 19 depressives before and after ECT. Regional and mean hemispheric flow were normal before and decreased by about 10% after ECT. Responders to ECT had higher flows before treatment. Risberg (1980) studied rCBF at rest by the same method but without pCO_2 correction in 70 patients with affective disorders. He showed normal mean levels and normal flow distributions, in contrast to organic dementia where rCBF was generally decreased (with parietal decrease on the Alzheimer group and frontal abnormalities in the Pick group). The same authors (Silfverskiöld *et al*, 1979) studied rCBF before and after ECT in 13 additional patients with depression and also found a normal rCBF before and a reduced rCBF after ECT, as did Johanson *et al* (1979). In none of these reports was any information available on treatment before the study or on the characteristics of the depressed patients studied. Mathew *et al* (1980) tested rCBF in 13 patients with major depression after two weeks of wash-out and 13 matched controls. Multidetectors were used and data were not corrected for pCO_2. The rCBF was lower bilaterally in depression and a significant negative correlation was found between rCBF and scores on the Hamilton Rating Scale for Depression. Gustafson *et al* (1981) studied 46 depressed patients and compared them with demented patients and normal subjects. There was a significantly lower hemispheric mean blood flow in depressed patients when compared with controls, but, when corrections for age and pCO_2 were made, the flow differences became less obvious.

Gur *et al* (1982) pointed out the importance of sex and handedness on the hemispheric flow asymmetry during cognitive activity. The rCBF increased during cognitive tests and was greater in the left hemisphere during the performance of verbal tasks, while it was greater in the right hemisphere during the spatial tasks. Women and left-handed subjects had a lesser degree of hemispheric cognitive specialisation. Gur *et al* (1984) then measured rCBF by the xenon-inhalation technique with 16 collimated sodium-iodide crystal detectors in 14 right-handed medicated depressives and in 25 matched controls, both at rest and after cognitive tests. Blood flows while resting did not differ in the two groups, although depressed male patients had lower than normal resting flows. Depressed female patients had higher than normal flows in all conditions, while for men, flows increased to normal during cognitive activity.

TABLE I

Demographic and clinical characteristics of the sample and mean (s.d.) regional cerebral blood flow

	Number	Mean age (s.d.): years	Sex men	Sex women	Mean Hamilton score (s.d.)	Regional cerebral blood flow: ml/100g/min left	Regional cerebral blood flow: ml/100g/min right	Regional cerebral blood flow: ml/100g/min total
Depressed patients	38	39.3 (13.1)	17	21	26.9 (5.6)	49.7 (10.5)	49.4 (10.2)	99.2 (20.6)
Controls	16	44.7 (24.2)	8	8	–	48.8 (9.4)	49.5 (9.7)	98.3 (19.1)
RDC subgroups of depressed patients								
Primary	25	39.9 (13.3)	12	13	25.8 (5.4)	47.7 (10.6)	47.6 (10.5)	95.3 (21.1)
Secondary[1]	13	38.2 (13.3)	5	8	29.2 (5.4)	53.6 (9.4)	53.0 (8.9)	106.6 (18.3)
Endogenous	30	39.4 (13.5)	15	15	27.5 (5.2)	50.3 (11.0)	50.0 (10.7)	100.3 (21.6)
Non-endogenous	8	38.9 (12.8)	2	6	24.8 (6.7)	47.8 (8.3)	47.2 (8.6)	105.0 (16.8)
Unipolar	30	38.8 (13.2)	11	19	26.8 (5.8)	50.4 (10.4)	49.8 (10.0)	100.2 (20.4)
Bipolar	8	41.4 (13.4)	6	2	27.5 (4.7)	47.2 (10.9)	48.0 (11.5)	95.2 (22.4)
Subgroups of depressed patients following biological tests								
REM latency								
≤50 min	18	41.5 (14.3)	7	11	25.7 (3.9)	52.4 (10.9)	52.4 (10.4)	104.8 (21.7)
>50 min	15[2]	35.5 (10.9)	7	8	27.7 (7.5)	47.5 (9.0)	47.1 (8.9)	94.6 (18.0)
Dexamethasone suppression								
positive	15	42.4 (14.3)	4	11	27.5 (5.3)	53.1 (10.6)	52.8 (10.2)	105.9 (20.8)
negative	15[2]	41.5 (12.8)	10	5	25.3 (5.3)	48.1 (11.2)	47.9 (11.0)	96.0 (22.2)
REM latency								
⩽50 min plus dexamethasone suppression	8	40.1 (14.7)	1	7	25.0 (3.9)	58.0 (9.1)	57.2 (18.5)	115.2 (17.6)
>50 min and no dexamethasone suppression	5[2]	35.2 (9.0)	4	1	23.6 (7.6)	50.5 (14.3)	49.3 (13.8)	99.8 (28.1)
Subgroups according to Newcastle scale								
Endogenous	11	43.3 (16.3)	5	6	27.7 (5.5)	46.7 (12.3)	46.6 (11.6)	93.3 (23.9)
Non-endogenous	26[2]	38.2 (11.6)	11	15	26.3 (5.5)	51.2 (9.7)	50.8 (9.7)	101.9 (19.4)

1. Not secondary to alcoholism, drug abuse, or other psychotic disorder.
2. Data missing for some patients.

TABLE II

Left–right differences in blood flow in slice 3

	Total number	*Mean (s.e.m.) sums of differences in outer rim: integral from 64 angular positions, ml/100 g/min*						*Number of peaks: left–right differences*[1]		
		all subjects			*right-handed subjects only*					
		n	*asymmetry*	P	n	*asymmetry*	P	*0*	*1*	*≥2*
Depressed patients	38	36	431 (182)		27	423 (228)		22	5	11
Controls	16	14	588 (196)	0.621	11	603 (245)	0.596	12	2	2
RDC subgroups of depressed patients										
Primary	25	23	410 (237)		17	295 (297)		16	3	6
Secondary	13	13	467 (291)	0.874	10	643 (362)	0.433	6	2	5
Endogenous	30	29	407 (215)		21	352 (280)		15	5	10
Non-endogenous	8	7	529 (307)	0.777	6	672 (320)	0.535	7	0	1
Unipolar	30	29	621 (200)		22	648 (244)		16	5	9
Bipolar	8	7	−359 (302)	0.019[2]	5	−564 (390)	0.023[2]	6	0	2
Subgroups of depressed patients following biological tests										
REM latency										
≤50 min	18	17	425 (272)		15	425 (307)		11	3	4
>50 min	15	14	421 (272)	0.991	9	334 (350)	0.838	9	1	5
Dexamethasone suppression										
positive	15	15	491 (298)		11	505 (407)		8	3	4
negative	15	14	653 (267)	0.660	11	589 (294)	0.855	11	1	3
REM latency										
≤50 min plus dexamethasone suppression	8	8	621 (484)		7	587 (557)		4	2	2
>50 min and no dexamethasone suppression	5	5	1133 (345)	0.362	3	1000 (163)	0.576	4	0	1
Subgroups according to Newcastle scale										
Endogenous	11	10	−147 (377)		7	−366 (518)		5	1	5
Non-endogenous	26	25	686 (198)	0.026[2]	19	746 (233)	0.021[2]	16	4	6

1. Exceeding 10 ml/100 g/min/pixel.
2. Results taken as being statistically significant

Uytenhoef *et al* (1983) found left frontal hypervascularisation in 16 major depressives compared with controls and minor and euthymic bipolar patients. Wash-out was for 15 days except for benzodiazepines and lithium. However, these authors did not note handedness and did not make the $p\mathrm{CO}_2$ correction during the inhalation of xenon. Silfverskiöld & Risberg (1989) compared rCBF measured by xenon-133 SPECT in 33 depressed in-patients and 30 manic patients. Not all of them were free of medication and handedness was not noted. They showed that patients with depression or mania had a normal rCBF.

The method used in our study suppressed the cross-talk artefact inherent in the use of the multidetector, as the hemispheres were viewed simultaneously. A correction was made for $p\mathrm{CO}_2$. All the patients were assigned RDC subgroups of affective disorder, they all underwent drug wash-out, and we took account of handedness. Smoking habits were not investigated. Under these controlled circumstances, global right and left hemispheric flow did not differ between major depressed patients and normal subjects, in agreement with studies by Risberg (1980), Gur *et al* (1984), and Silfverskiöld & Risberg (1989).

When we considered only the cortical CBF, we found a significant left–right asymmetry, both for bipolar patients and patients with an endogenous depression according to the Newcastle scale, despite the small number of subjects in each category. The same results were documented for right-handed subjects only. These results suggest a relative hypoperfusion of the dominant hemisphere in bipolar patients and in patients with the endogenous subtype of depression.

Acknowledgements

We wish to thank Professor Flor-Henry for his critical comments on the manuscript. This work was supported in part by funds provided by the Ministère de la Politique Scientifique (Sciences de la vie – contrat BIO/04).

References

Alexander, S. C., Wollman, H., Cohen, P. J., *et al* (1964) Cerebrovascular response to $P_a\mathrm{CO}_2$ during halothane anesthesia in man. *Journal of Applied Physiology*, **19**, 561–565.

Berman, D. S., Garcia, E. V. & Maddahi, J. (1981) Thallium-201 myocardial scintigraphy in the detection and evaluation of coronary artery disease. In *Clinical Nuclear Cardiology* (eds D. S. Berman & D. T. Mason) pp. 49–106. London: Academic Press.

Carney, M. W. P., Roth, M. & Garside, R. F. (1965) The diagnosis of depressive syndromes and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 659–674.

Caroll, B. J., Curtis, G. C. & Mendels, J. (1976) Neuroendocrine regulation in depression. II : Discrimination of depressed from non-depressed patients. *Archives of General Psychiatry*, **33**, 1051–1058.

Celsis, P., Goldman, T., Henriksen, L., *et al* (1981) A method for calculating regional cerebral blood flow from emission computed tomography of inert gas concentration. *Journal of Computer Assisted Tomography*, **5**, 641–645.

D'Elia, G. & Perris, C. (1973) Cerebral functional dominance and depression. *Acta Psychiatrica Scandinavica*, **49**, 191–197.

Flor-Henry, P. (1976) Lateralized temporal-limbic dysfunction and psychopathology. *Annals of the New York Academy of Sciences*, **280**, 777–795.

Gruzelier, J. & Flor-Henry, P. (1979) *Hemispheric Asymmetries of Function in Psychopathology*. Amsterdam: Biomedical Press.

Gur, R. C., Gur, R. E., Obrist, W. D. *et al* (1982) Sex and handedness differences in cerebral blood flow during rest and cognitive activity. *Science*, **213**, 659–661.

Gur, R. E., Skolnick, B. E., Gur, R. C., *et al* (1984) Brain function in psychiatric disorders. II. Regional cerebral blood flow in medicated unipolar depressives. *Archives of General Psychiatry*, **41**, 695–699.

Gustafson, L., Risberg, J. & Silfverskiöld, P. (1981) Regional cerebral blood flow in organic dementia and affective disorders. In *Electroneurophysiology and Psychopathology. Advances in Biological Psychiatry* (vol. 6). (Eds C. Perris, D. Kemali & L. Vacca), pp. 109–116. Basel: Karger.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

Johanson, M., Risberg, J. Silfverskiöld, P., *et al* (1979) Regional cerebral blood flow related to acute memory disturbance following electroconvulsive therapy on depression. *Acta Neurologica Scandinavica*, **60** (suppl. 72), 534–535.

Kanno, I. & Lassen, N. (1979) Two methods for calculation of regional cerebral blood flow from emission computed tomography of inert gas concentration. *Journal of Computer Assisted Tomography*, **3**, 71–76.

Kupfer, D. J., Foster, F. G., Coble, P., *et al* (1978) The application of the EEG sleep for the differential diagnosis of affective disorders. *American Journal of Psychiatry*, **135**, 69–74.

Lassen, N. & Rommer, P. (1986) Cerebral blood flow tomography by single photon emission computerized tomography using ^{133}Xenon in cerebrovascular disease. In *Monograph on Atherosclerosis*, vol. 14 (eds A. Ventura, G. Crepaldi & V. Senin), pp. 71–82. Basel: Karger.

Lipsey, J. R., Robinson, R. G., Pearlson, G. D., *et al* (1983) Mood change following bilateral hemisphere brain injury. *British Journal of Psychiatry*, **143**, 266–273.

Lishman, W. A. (1968) Brain damage in relation to psychiatric disability after head injury. *British Journal of Psychiatry*, **144**, 373–410.

Mathew, R. J., Meyer, J. S., Francis, D. J., *et al* (1980) Cerebral blood flow in depression. *American Journal of Psychiatry*, **137**, 1449–1450.

Mendlewicz, J., Kerkhofs, M., Hoffmann, G., *et al* (1984) Dexamethasone suppression test and REM sleep in patients with major depressive disorders. *British Journal of Psychiatry*, **45**, 383–388.

Nie, N. H., Hull, C. H., Jenkins, J. G., *et al* (1975). *Statistical Package for the Social Sciences* (2nd edn). New York: McGraw-Hill.

Obrist, W. D., Thompson, H. K., Wang, H. S., *et al* (1975) Regional cerebral blood flow estimated by ^{133}Xenon inhalation. *Stroke*, **6**, 245–256.

Oldfield, R. C. (1971) The assessment and analysis of handedness: the Edinburgh Inventory. *Neuropsychologia*, **9**, 97–113.

Rechtschaffen, A. & Kales, A. (1968) *A Manual of Standardised Terminology, Techniques and Storing System for Sleep Stages of Human Subjects*. Washington, DC: USPHS Government Printing Office.

RISBERG, J. (1980) Regional cerebral blood flow measurements by ^{133}Xenon-inhalation: methodology and applications in neuropsychology and psychiatry. *Brain and Language*, 9, 9–34.

ROOTWELT, K., DYBEVOLD, S., NYBERG-HANSEN, R., *et al* (1986) Measurement of cerebral blood flow with ^{133}Xenon inhalation and dynamic single photon emission computer tomography. Normal values. *Scandinavian Journal of Clinical Laboratory Investigations*, 46, (suppl. 184), 97–105.

SACKEIM, H. A., GREENBERG, M. S., WEINAN, A. L., *et al* (1982) Hemispheric asymmetry in the expression of positive and negative emotions. *Archives of Neurology*, 39, 210–218.

SILFVERSKIÖLD, P., GUSTAFSON, L., JOHANSON, M., *et al* Regional cerebral blood flow related to the effect of electroconvulsive therapy in depression. In *Biological Psychiatry Today* (eds J. Obiols, C. Ballus, E. Gonzalez, *et al*), pp. 1178–1183. Amsterdam: Elsevier.

—— & RISBERG, J. (1989) Regional cerebral blood flow in depression and mania. *Archives of General Psychiatry*, 46, 253–259.

SPITZER, R. L., ENDICOTT, J. & ROBBINS, E. (1975) *Research Diagnostic Criteria for a Selected Group of Functional Disorders* (2nd edn). New York: State Psychiatric Institute.

UYTENHOEF, P., PORTELANGE, P., JACQUY, J., *et al* (1983) Regional cerebral blood flow and lateralized hemispheric dysfunction in depression. *British Journal of Psychiatry*, 143, 128–132.

V. Delvenne, MD, *Assistant, Department of Psychiatry, Erasme Hospital*; F. Delecluse, MD, *Assistant, Department of Nuclear Medicine, Erasme Hospital*; Ph. P. Hubain, MD, *Resident, Department of Psychiatry, Erasme Hospital*; A. Schoutens, MD, PhD, *Professor and Head, Department of Nuclear Medicine, Erasme Hospital*; V. De Maertelaer, PhD, *Professor, IRIBHN Statistical Unit, Free University of Brussels, Brussels, Belgium*; *J. Mendlewicz, MD, PhD, *Professor and Head, Department of Psychiatry, Erasme Hospital, University Clinics of Brussels, Free University of Brussels, 808 route de Lennik, 1070 Brussels, Belgium*

*Correspondence

British Journal of Psychiatry (1990), **157**, 366–372

Dichotic Perception and Memory Following Electroconvulsive Treatment for Depression

KARL M. WILLIAMS, WILLIAM G. IACONO, RONALD A. REMICK and PATRICK GREENWOOD

Verbal and visuospatial memory and dichotic listening performance were examined in 15 acutely depressed patients with no history of ECT, 17 depressed patients currently in remission, 15 remitted depressed patients who had received ECT six months or more in the past, and 20 normal controls. The neuropsychological functioning of an additional group of 10 acutely depressed patients was also studied before and two weeks after ECT. The results revealed some evidence of logical and autobiographical memory impairment two weeks following ECT, but no evidence that ECT impaired dichotic listening ability. Rather, a normalisation of hemispheric laterality was apparent on the dichotic listening task following ECT and the concomitant relief from depression. There was also no evidence of cognitive dysfunction on any task in individuals who were tested six months or more following their last ECT treatment.

Although there have been a great many studies examining cognitive ability following electroconvulsive therapy (ECT), substantial variability in the quality, methodology, and outcome of these investigations has complicated their interpretation. Hence, it has not been possible to attain a consensus regarding possible deleterious effects of ECT. Several reports have suggested that no prolonged cognitive deficits exist after ECT (e.g. Cronholm & Molander, 1964; Brunschwig *et al*, 1971; Small, 1974; Squire & Chace, 1975; Squire *et al*, 1981; Weeks *et al*, 1980; Frith *et al*, 1983). However, most studies have either indicated that residual neuropsychological impairment follows ECT (e.g., Halliday *et al*, 1968; Bidder *et al*, 1970; Cronin *et al*, 1970; Goldman *et al*, 1972; Temper *et al*, 1973; Squire *et al*, 1976; Freeman *et al*, 1980; Steif *et al*, 1986), or they have yielded mixed or inconclusive data concerning protracted deficits after ECT (Cronholm & Ottoson, 1961, 1963; Bilikiewicz & Krzyzowski, 1964; Miller, 1970; Squire *et al*, 1979; Freeman & Kendell, 1980; Daniel *et al*, 1983). Our review of the literature indicated that of 43 studies that contained information on long-term (mean 26.5 months post-treatment) cognitive functioning after ECT, 19 studies included data indicative of protracted cognitive deficits. Although a wide variety of intelligence, mental status, and memory tests have been employed in previous ECT research, an alternative but seldom used approach to the investigation of ECT-related deficits involves examining information-processing capability directly, using, for example, dichotic listening tasks.

Dichotic listening (DL), introduced by Broadbent (1956), occurs when different auditory stimuli are presented simultaneously to the left and right ears. It has been found (e.g., Broadbent & Gregory, 1964; Kimura, 1961*a*, 1963) that in dichotic perception tasks normal individuals usually exhibit right-ear superiority in the detection of verbal material and left-ear superiority in the detection of non-verbal material. Trauma to the brain in the vicinity of the temporal lobe in the right hemisphere has been found to result in deficits in the perception of material presented to the left ear. This results in an exaggeration of the relative superiority of the right ear in tasks that involve dichotic perception of verbal material and diminishes the superiority of the left ear in the perception of non-verbal material. Lesions to the left hemisphere of the brain have a more variable effect upon dichotic perception. Left-sided injury has been found to result in bilateral impairment in the perception of verbal material and to exert varied effects upon the perception of non-verbal material (Kimura, 1961*b*; Schulhoff & Goodglass, 1969; Netley, 1972; Zurif & Ramier, 1972; Efron *et al*, 1977; Knellinger, 1981; Efron & Crandall, 1983).

Of particular relevance to ECT are the investigations that have used DL performance as an index of cerebral and especially temporal lobe dysfunction. This relevance is derived from the fact that although there is variability in the actual placement of the electrodes during the ECT procedure, the usual North American clinical application results in the discharge of electricity over or near the right temporoparietal area of the brain. The results of two previous studies which examined the dichotic perceptual ability of patients who had received unilateral ECT suggest that dichotic perception tasks may be sensitive to ECT-induced cognitive changes (Altman *et al*, 1979; Moscovitch *et al*, 1981).

The present study was conducted to help determine whether cognitive dysfunction follows ECT. Two

TABLE I
Demographic characteristics of subjects

Group	*No. of subjects* *female*	*male*	*Age: years* *mean*	*s.d.*	*range*	*Years of education* *mean*	*s.d.*
Current ECT	7	4	56.8	11.5	36–66	12.18	1.68
Depressed, non-ECT	11	4	40.8	13.0	24–68	12.53	2.72
Past ECT	9	6	48.3	12.0	31–65	12.47	2.72
Remitted, non-ECT	14	3	46.2	11.2	26–64	14.06	2.56
Normal	13	7	41.6	12.9	22–63	13.20	3.32

experimental groups, one consisting of patients evaluated before and after ECT and the other of individuals who had received ECT at least six months previously, were used. These groups were compared to currently depressed patients not receiving ECT and remitted depressives who had never received ECT. The latter two groups provided an opportunity to examine the dependent variables in individuals with a history of depression but not of convulsive treatment. Finally, a group of normal volunteers were included to assess task performance in persons with a history of neither depression nor ECT.

This study extended previous investigations of the neuropsychological performance of ECT-treated patients by using a standardised and reliable audiological test of dichotic perception. Various tests of memory were also included in the protocol because most studies of the cognitive effects of ECT have focused on possible memory dysfunction resulting from the procedure. It was hypothesised that any cognitive deficits present after ECT would be manifest by an abnormal exaggeration of the normal right-ear advantage on the DL test and by diminished performance on the tests of memory.

Method

Subjects

Fifty-eight patients in treatment at a major metropolitan hospital for a primary complaint of depression and twenty normal control subjects without history or symptoms of mental disorder gave informed consent to participate in this study. All subjects were right-handed, defined operationally as a score of at least 35, out of a possible 50, on the Edinburgh checklist criteria (Oldfield, 1971). On the basis of their clinical history, current status, and treatment, depressed patients were assigned to one of four groups: (a) depressed, non-ECT ($n = 15$); (b) remitted, non-ECT ($n = 17$); (c) current ECT (in treatment with right unilateral ECT, $n = 11$); (d) past ECT (received right unilateral ECT not less than six months prior to testing, $n = 15$). The normal control group consisted of individuals from local offices of the Unemployment Insurance Commission who were paid for their participation in the study.

Diagnoses were made independently by two clinicians, one of whom (RAR) conducted a clinical interview. The other (KMW) administered a modified version of the Diagnostic Interview Schedule (DIS; National Institute of Mental Health, 1981) and the Beck Depression Inventory (BDI; Beck *et al*, 1961) on the day the dichotic listening and memory tasks were given.

All patients, whether in remission or currently depressed, received a primary diagnosis of major affective disorder with depression according to the criteria specified in DSM-III (American Psychiatric Association, 1980). Thirty-nine of the patients had received a diagnosis of recurrent major depression (unipolar) and 19 of bipolar depression. Forty-six of the patients were receiving antidepressant medication or lithium carbonate when tested and 12 had not received medication for at least one month before testing. Subject characteristics are summarised in Tables I and II.

ECT characteristics

ECT was administered as sinusoidal current (Medcraft B-24 machine) for 0.5 to 1.0 seconds at 110 to 170 volts. A right non-dominant unilateral electrode placement as described by d'Elia (1970) was used. Standard anaesthesia (methohexital, succinolcholine) was administered before ECT. No preoperative atropine was given. In the majority of cases, treatments were given three times weekly. Seizure duration was monitored by the cuff method (Fink & Johnson, 1982) with all attempts to keep seizure duration between 25 and 40 seconds. Patients received between eight and 12 treatments in their course of ECT.

Audiometry

All subjects were tested audiometrically and for accuracy of dichotic perception in a Tracoustics RS257DS double-walled, sound-attenuating booth. Auditory stimuli were conveyed to the subject by means of Telephonics TDH-39 headphones in circumaural cushions. Audiometric readings of stimulus frequency and intensity level were made on a Madsen OB77 audiometer. Dichotic perception test instructions and stimuli were pre-recorded on cassette tape by Auditec of St Louis, Missouri (Katz, 1973, 1985). Dichotic perception was assessed by means of the staggered spondaic word (SSW) test, a standardised DL test that is commonly used in clinical audiometry and that has been the subject of much research with a variety of normal and

TABLE II
Clinical characteristics of subjects

Group	*Medicated?*		*Diagnosis*		*Beck Depression Scale score*	
	yes	*no*	*UP*[1]	*BP*[2]	*mean*	*s.d.*
Current ECT						
pre-treatment	10	1	8	3	29.64	10.93
post-treatment	11	0	8	3	12.20	11.46
Depressed non-ECT	12	3	10	5	29.20	6.20
Past ECT	13	2	9	6	8.00	10.18
Remitted non-ECT	11	6	12	5	7.47	6.50
Normal	0	20	0	0	4.45	4.58

1. UP = Unipolar affective disorder (DSM-III criteria for recurrent depression).
2. BP = Bipolar affective disorder (DSM-III criteria).

clinical populations (e.g., Katz *et al*, 1963; Spitzer & Ventry, 1980; Arnst, 1981; Wetherby *et al*, 1981). The SSW test is a dichotic word perception task in which two spondaic words (e.g., upstairs; downtown) are presented, one to each ear. The words are staggered in time so that the second syllable of the first word occurs simultaneously with the first syllable of the second word. A series of 20 word pairs is presented in the test. The task of the subject is to repeat each of the pairs after it has been presented; the failure to do so correctly constitutes an error.

Administration and scoring of the SSW test followed the standard procedures employed routinely in clinical audiological evaluations (Arnst, 1982). To ensure correct administration of the SSW test, an audiometric examination (e.g. Hughson & Westlake, 1944) of the subject was undertaken before testing. This examination provided the following information for each subject: (1) pure-tone threshold; (2) three-frequency pure-tone average, defined as the mean intensity threshold for tones that were presented at frequencies of 500, 1000 and 2000 Hz; and (3) speech reception threshold, defined as the lowest intensity at which 50% of the spondees were repeated correctly. This information was then used to determine the speech discrimination score (SDS) of the subject. This score, expressed as a percentage error, indicated the accuracy with which the subject repeated a series of words presented 30 dB above his or her speech reception threshold. The SSW test stimuli were presented to the subject at a level of intensity that was 50 dB above the three-frequency pure tone average. The audiological examination and the SSW test were administered by a qualified clinical audiologist (PG) and scored (as specified by Arnst, 1981) such that the percentage of errors for each subject for the left and right ear was determined after correcting for the SDS. This was accomplished by subtracting the percentage SDS error for each ear from the raw percentage of errors for each ear. By correcting each participant's score in this fashion, subjects were not penalised for any hearing difficulties they may have had.

Memory

Memory was tested with a battery of tasks selected to provide an index of both anterograde and retrograde memory. Tests of anterograde functioning examined immediate and delayed recall of verbal and visuospatial material. Retrograde memory was evaluated with a test of autobiographical memory. All of the commercially available tests were administered and scored following the instructions outlined in the test manuals. Alternate forms of the tests (except recurring figures and autobiographical recall) were used for subjects who were tested twice. The memory tests employed were as follows.

(a) *Recurring figures test* (Kimura, 1963), a test that is sensitive to right temporal lobe dysfunction (Kimura, 1963; Rixecker & Hartje, 1980). Subjects are asked to recognise 40 figures that are embedded in a deck of 100 cards.

(b) *Digit span* (Wechsler, 1945, 1981), considered to be a test of short-term memory, or immediate auditory memory, concentration, attention, and the ability to shift thought patterns from digits forward to digits backward.

(c) *Benton visual retention test* (Benton, 1974) a test of the ability to recall and reproduce from memory geometric designs presented on ten cards. Forms D and E (administration D) were used.

(d) *Logical memory*, from the Wechsler Memory Scale (Wechsler, 1945), considered to be a test of the ability to recall logical verbal material. This test required subjects to recall details of two stories read to them after delays of five and three minutes respectively, during which time they completed questionnaires. Forms I and II were used.

(e) *Autobiographical memory*, a 32-item questionnaire designed specifically for the present research. The questionnaire was patterned after a scale used by Squire *et al* (1981) and was intended to assess the ability of the subject to recall material of an autobiographical nature. Subjects were required to recall both objective and subjective information either from their distant (over three years ago) or recent (within the last three years) past. The scoring indicated how many personal memories the subject could recall. It was not possible to verify the truth of the recalled memories. Sample questions were 'What did you do during your 1984 summer vacation?' and 'What was your favourite TV programme during the 1970's?'

The above tests, in addition to the BDI, DIS and the Edinburgh Handedness Checklist, were administered on the same day that the audiometric assessment and the SSW test were conducted. All subjects except those in the current-ECT group were tested once. The current-ECT patients were tested twice, once a few days before starting ECT and again approximately two weeks after the conclusion of the ECT series.

Results

Applications of analysis of variance (ANOVA) and the χ^2 statistic revealed no differences among groups in educational attainment, proportion of bipolar depressives, or sex composition. However, an ANOVA based on age indicated that the groups differed in age ($F(4,73) = 3.61$, $P < 0.01$). *Post hoc* comparisions revealed that the differences were attributable to the fact that the subjects in the current-ECT group were significantly older than those in the depressed non-ECT and normal groups, although the past-ECT group did not differ in age from the other groups. Accordingly, in the among-group analyses of the neuropsychological data that follow, age was used as a covariate.

As expected, the groups differed in level of depression on the BDI, both before and after the subjects in the current-ECT group had undergone treatment ($F(4,73) = 39.61$, $P < 0.001$ and $F(4,72) = 26.10$, $P < 0.001$, respectively). Multiple comparisons revealed that before commencing treatment, the subjects in the current-ECT and depressed non-ECT groups did not differ from one another in level of depression, but both groups of patients were significantly more depressed than those in all of the other groups. After treatment the depressed non-ECT group was significantly more depressed than all the other groups, including the current-ECT group which showed a level of depression similar to that in the other groups (see Table II).

Dichotic listening

ECT patients before and after treatment

The dichotic listening performance of the current-ECT patients was evaluated by conducting a 2(ears) × 2(times) repeated measures ANOVA. Neither the main effect for ears nor time was significant. The interaction effect, however, was significant ($F(1,9) = 5.21$, $P < 0.05$), and indicated that right-ear performance improved significantly ($F(1,9) = 8.96$, $P < 0.5$) while left ear performance was unchanged ($F(1,9) = 0.52$).

Group comparisons

Table III shows the results of the SSW test for all groups. It was hypothesised that comparison of right- and left-ear performance on the SSW task would differentiate those who had received ECT in the past from the various control subjects who had not. To test this prediction, the right- and left-ear dichotic perceptual scores were compared for the subjects in all but the current-ECT group by calculating a 2(ears) × 4(groups) ANCOVA with repeated measures on the first factor. The main effect for ears was significant ($F(1,62) = 18.46$, $P < 0.001$), but the main effect for groups ($F(3,62) = 1.15$) and the ear by group interaction ($F(3,62) = 1.35$) were not. Paired *t*-tests comparing left- and right-ear performance for each group were calculated; all four tests confirmed that right ear errors were less frequent than left ear errors. The results confirmed that all of the groups performed in a similar fashion and demonstrated right-ear superiority; indeed, all generated mean scores that fall within normal limits (Katz, 1973).

Memory

ECT patients before and after treatment

To test the hypothesis that the current-ECT patients would show a decrement in performance following ECT, the memory performance of the current-ECT patients was contrasted before and after they had received ECT by

TABLE III

Mean values (s.d.) of memory and dichotic performance by group

Test	*Current ECT*		*Depressed*	*Past ECT*	*Remitted*	*Normal*
	Pre-ECT	*Post-ECT*	*non-ECT*		*non-ECT*	
SSW left[1]	5.91 (8.14)	5.70 (10.46)	4.27 (5.42)	4.27 (3.95)	1.50 (2.37)	3.40 (3.35)
SSW right[1]	5.82 (9.38)	2.10 (5.92)	1.40 (2.75)	0.00 (5.13)	0.62 (2.75)	1.20 (1.76)
Autobiographical memory[2]	26.09 (3.11)	23.60 (5.02)	28.73 (3.45)	28.87 (2.44)	29.82 (2.00)	29.85 (2.75)
Recurring figures[3]	19.18 (9.32)	20.50 (9.37)	24.87 (6.85)	22.07 (7.78)	26.12 (7.02)	26.70 (8.26)
Digits backward[4]	4.36 (1.12)	4.50 (1.35)	4.53 (1.12)	4.53 (0.92)	5.18 (1.47)	4.70 (0.73)
Logical memory[5]	7.36 (3.29)	5.60 (3.78)	9.00 (3.25)	8.87 (4.08)	10.65 (3.69)	12.75 (2.77)
Benton visual retention[6]	3.00 (2.57)	3.78 (2.82)	6.87 (2.55)	5.93 (2.66)	7.06 (2.77)	7.95 (1.82)

1. Percent error corrected for each subject's hearing ability. Unlike the memory tasks, a high score on this test reflects poor performance.
2. Number of memories recalled.
3. Number of correct identifications minus the number incorrectly identified as having been seen before.
4. Number of digits repeated.
5. Number of ideas recalled from two memory passages.
6. Number of correct reproductions.

conducting a series of correlated *t*-tests. These tests were based on 10 subjects because one patient in this group was unavailable after treatment. As can be seen in Table III, test performance actually improved slightly across occasions for the digit span, recurring figures, and Benton tasks, but autobiographical memory ($t(9) = -2.01$, $P < 0.05$) and logical memory ($t(9) = -1.92$, $P < 0.05$) performance were significantly worse following treatment.

Group comparisons

A multivariate analysis of covariance (MANCOVA) was conducted to compare the memory capability of the groups after the current-ECT patients had finished treatment. The significant multivariate effect ($F(24,228) = 1.93$, $P < 0.01$) was further evaluated by conducting univariate ANCOVAs which indicated that the groups differed significantly on logical ($F(4,72) = 5.05$, $P < 0.01$) and autobiographical ($F(4,72) = 5.97$, $P < 0.001$) memory. *Post hoc* comparisons revealed that these results were attributable to the patients in the current-ECT group, after treatment, having the least number of autobiographical and logical memories.

Medication effects

There were no significant differences between medicated and unmedicated depressive subjects in their performance on the memory and dichotic listening tasks.

Discussion

Dichotic listening

A major innovation of the present study involved the use of a standardised dichotic listening task commonly employed in clinical audiometry that is sensitive to various types of brain dysfunction (see Arnst & Katz, 1982). It was hypothesised that ECT would lead to an exaggeration of normal right-ear superiority, perhaps manifest by an increase in left-ear errors. The results, however, revealed a slight drop in left-ear error and a significant drop in right-ear error in the current-ECT subjects. Before ECT, there was no difference in error rate between ears, a result that is consistent with other reports that found dichotic listening deficits in acutely ill patients with major affective disorder (Bruder *et al*, 1981; Johnson & Crockett, 1982; Lisman *et al*, 1978; Moscovitch *et al*, 1981; Yozawitz *et al*, 1979). The significant improvement in the right-ear dichotic listening performance of the current-ECT subjects after treatment suggests a normalisation of hemispheric functioning after ECT, rather than a change in functioning attributable to right-hemisphere dysfunction. Following conclusion of treatment and clinical improvement, the right-ear error rate for the ECT subjects was within the normal range and consistent with that of the other subjects (see Table III). This pattern of results is similar to that reported by Moscovitch *et al* (1981) who concluded that the alleviation of depression following ECT may have had a normalising effect on dichotic listening in a small group of seven depressed patients.

The dichotic listening findings from the past-ECT group support the above conclusion that ECT does not lead to a DL performance deficit. The past-ECT subjects showed a normal pattern of listening performance, thereby indicating that there is no significant residual effect of ECT on dichotic performance six or more months following treatment.

Memory tasks

Five univariate statistics were calculated to determine if a decrement in memory performance existed following ECT. Two of these tests generated significant results despite the small sample size of the current-ECT group and associated lack of power to detect significant effects. Indeed, a possible concern with these analyses is that clinically meaningful performance decrements may exist following ECT, but that they were not statistically significant because only a small group of patients was studied. This possibility is not supported by the data, however, which shows that for the three memory tests that failed to yield significance, memory performance actually improved slightly following ECT.

Do patients who have recently completed ECT show a memory deficit relative to other subjects who have not? Our analyses showed that the current-ECT group performed more poorly after ECT than the other subjects on two of the five memory tests, autobiographical memory and logical memory. The two memory test-analyses complement and reinforce one another because in both cases, memory deficits were identified in these same two tests, but were not found in the other three memory tasks. These findings, that recent administration of ECT leads to impaired performance on these two tests, and that patients recently administered ECT are impaired on the same two tests compared with other groups of depressives and normal subjects, provide compelling evidence that autobiographical and logical memory are impaired during the two weeks following ECT.

The deficits observed in the current-ECT group following treatment existed despite the fact that for one of the tests (autobiographical memory), the identical scale was administered on both occasions. This observation of post-ECT deficits in autobiographical memory is consistent with that of previous work in this area (e.g. Squire *et al*, 1981). In addition, the poor performance of these subjects cannot be attributed to depressed mood. The clinically depressed non-ECT patients did not show a similar deficit, and the current-ECT subjects were substantially less depressed following ECT. The average level of depression of the subjects in the current-ECT group, as reflected by BDI scores, was 29.64 before treatment and only 12.20 after treatment.

Although all the memory tests require the retrieval of memories, the logical and autobiographical memory tasks place great demand on retrieval processes. The only other test that emphasises memory retrieval, the Benton, did not show significant impairment. However, subjects were asked to draw the Benton figures after only a 15-second delay during which there was no interference from other tasks. By contrast, the two logical memory passages were presented with delays of five and three minutes, and during the delay interval, subjects were required to complete questionnaires. The recurring figures test, on which current-ECT patients performed well, also involves a delay between stimulus presentation and the memory test. However, this is a recognition test. The fact that patients performed well on this test indicates that they were able to acquire new information because they could recognise it later. Another difference between the recurring figures and Benton tests and the autobiographical and logical memory tests is that the former involve visuospatial memory while the latter are concerned with verbal material. Hence, the nature of the information subjects were asked to remember could account for the different performance. Nevertheless, overall this pattern of results suggests that there is a problem with long-term memory retrieval of verbal material two weeks following ECT.

By including the past-ECT group it was possible to determine if ECT was associated with residual or protracted memory dysfunction. Since performance deficits were observed in the past-ECT patients, such problems may not persist six months or more after treatment. This failure to detect evidence of prolonged cognitive deficit is consistent with a number of reports from other investigators (see introduction).

Conclusion

Dichotic listening performance following ECT was not indicative of right hemisphere dysfunction, but there was some evidence of abnormal lateralisation on this task in the group of acutely depressed patients scheduled for ECT. The results of the present investigation support the notion that the retrieval of verbal material from long-term memory is impaired two weeks following right unilateral ECT. Memory deficits were not detected in visuospatial performance based on tasks that required recognition of figures or reproduction of designs after a short delay. Immediate auditory memory and attention, as indexed by the digit span test, were also normal two weeks post-ECT. The results of the present study provided no evidence of long-term (that is, six months or more after treatment) deficits in cognitive functioning associated with ECT.

Acknowledgement

This research was supported in parts by grants from the Medical Research Council of Canada and the University of Minnesota Graduate School.

References

Altman, J. A., Balanor, L. J. & Deolin, V. L. (1979) Effects of unilateral disorder of the brain hemisphere function in man or directional hearing. *Neuropsychologia*, 17, 295–301.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Arnst, D. J. (1981) Errors on the staggered spondaic word (SSW) test in a group of adult normal listeners. *Ear & Hearing*, 2, 112–116.

—— (1982) Overview of the staggered spondaic word test and the competing environmental sounds test. In *Central Auditory Assessment: The SSW Test – Development and Clinical Use* (eds D. Arnst & J. Katz). San Diego: College Hill Press.

—— & Katz, J. (1982) *Central Auditory Assessment:The SSW Test – Development and Clinical Use*. San Diego: College Hill Press.

Beck, A. T., Ward, C. H., Mendelson, M., *et al* (1961) An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571.

Benton, A. L. (1974) *The Revised Visual Retention Test* (4th edn). New York: Psychological Corporation.

Bidder, T. E., Strain, J. J. & Brunschwig, L. (1970) Bilateral and unilateral ECT: follow-up study and critique. *American Journal of Psychiatry*, 127, 37–44.

Bilikiewicz, A. & Kryszoswki, J. (1964) Application of unilateral electric shocks in psychiatry. *Neurologia I. Neurochirurgia Psychiatria Polska*, 14, 663–669.

Broadbent, D. E. (1956) Successive responses to simultaneous stimuli. *Quarterly Journal of Experimental Psychology*, 8, 145–162.

—— & Gregory, M. (1964) Accuracy of recognition for speech presented to the right and left ears. *Quarterly Journal of Experimental Psychology*, 16, 359–360

Bruder, G. E., Sutton, S., Berger-Gross, P., *et al* (1981) Lateralised auditory processing in depression: dichotic click detection. *Psychiatry Research*, 4, 253–266.

Brunschwig, L., Strain, J. J. & Bidder, T. E. (1971) Issues in the assessment of post-ECT memory changes. *British Journal of Psychiatry*, 119, 73–74.

Cronholm, B. & Molander, L. (1964) Memory disturbances after electroconvulsive therapy. 5. Conditions one month after a series of treatments. *Acta Psychiatrica Scandinavica*, 40, 212–216.

—— & Ottoson, J. O. (1961) Memory functions in endogenous depression: before and after electroconvulsive therapy. *Archives of General Psychiatry*, 5, 101–107

—— & —— (1963) The experience of memory function after electroconvulsive therapy. *British Journal of Psychiatry*, 109, 251–258.

Cronin, D., Bodley, P., Potts, L., *et al* (1970) Unilateral and bilateral ECT: a study of memory disturbances and relief from depression. *Neurology, Neurosurgery, and Psychiatry*, 33, 705–713.

Daniel, W. F., Weiner, R. D. & Crovitz, H. F. (1983) Autobiographical amnesia with ECT: an analysis of the roles of stimulus wave form, electrode placement, stimulus, energy, and seizure length. *Biological Psychiatry*, **18**, 121–126.

D'elia, G. (1970) Unilateral electroconvulsive therapy. *Acta Psychiatrica Scandinavica* (suppl. 215), 5–98.

Efron, R. & Crandall, P. H. (1983) Central auditory processing II. Effects of anterior temporal lobectomy. *Brain & Language*, **19**, 235–253.

——, Dennis, M. & Yund, E. W. (1977) The perception of dichotic chords by hemispherectomized subjects. *Brain & Language*, **4**, 537–549.

Fink, M. & Johnson, L. (1982) Monitoring duration of ECT seizures: "cuff" and EEG methods compared. *Archives of General Psychiatry*, **39**, 1189–1191.

Freeman, C. P. L. & Kendell, R. E. (1980) ECT: I. Patients' experiences and attitudes. *British Journal of Psychiatry*, **137**, 17–25.

——, Weeks, D. & Kendell, R. E. (1980) ECT: II. Patients who complain. *British Journal of Psychiatry*, **137**, 17–25.

Frith, C. D., Stevens, M., Johnstone, E. C., *et al* (1983) Effects of ECT and depression on various aspects of memory. *British Journal of Psychiatry*, **142**, 610–617.

Goldman, H., Gomer, F. E. & Templer, D. I. (1972) Long-term effects of electroconvulsive therapy upon memory and perceptual-motor performance. *Journal of Clinical Psychology*, **28**, 32–34.

Halliday, A. M., Davison, K., Browne, M. W., *et al* (1968) A comparison of the effects on depression and memory of bilateral ECT and unilateral ECT to the dominant and nondominant hemispheres. *British Journal of Psychiatry*, **114**, 997–1012.

Hughson, W. & Westlake, H. D. (1944) Manual for program outline for rehabilitation of aural casualties both military and civilian. *Transactions; American Academy of Ophthalmology and Otolaryngology* (suppl.), **42**, 1–15.

Inglis, J. (1970) Shock, surgery, and cerebral asymmetry. *British Journal of Psychiatry*, **117**, 143–148.

Johnson, O. & Crockett, D. (1982) Changes in perceptual asymmetries with clinical improvement of depression and schizophrenia. *Journal of Abnormal Psychology*, **91**, 45–54.

Katz, J. (1973) *The SSW Test Manual*. Brentwood, MO: Auditec of St Louis.

—— (1985) *Handbook of Clinical Audiology* (3rd edn). Baltimore: Williams & Wilkins.

——, Basil, R. A. & Smith, J. M. (1963) A staggered spondaic word test for detecting central auditory lesions. *Annals Otology, Rhinology & Laryngology*, **72**, 908–917.

Kimura, D. (1961*a*) Cerebral dominance and the perception of verbal stimuli. *Canadian Psychology*, **15**, 166–171.

—— (1961*b*) Some effects of temporal lobe damage on auditory perception. *Canadian Psychology*, **15**, 156–165.

—— (1963) Right temporal lobe damage. *Archives of Neurology*, **8**, 264–271.

Lishman, W. A., Toone, B.K., Colbourn, C. J. *et al* (1978) Dichotic listening in psychotic patients. *British Journal of Psychiatry*, **132**, 333–341.

Miller, E. (1970) The effect of ECT on memory and learning. *British Medical Psychology*, **43**, 57–62.

Moscovitch, M., Strauss, E. & Olds, J. (1981) Handedness and dichotic listening performance in patients with unipolar endogenous depression who received ECT. *American Journal of Psychiatry*, **138**, 988–990.

National Institute Of Mental Health (1981) *NIMH diagnostic interview schedule: Version III* (DHHS Publication No. ADM-T-42-3). Washington, DC: US Government Printing Office.

Netley, C. (1972) Dichotic listening performance of hemisphericectomized patients. *Neuropsychologia*, **10**, 233–240.

Oldfield, R. C. (1971) The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, **9**, 97–113.

Rixecker, H. & Hartje, W. (1980) Kimura's recurring-figures test: a normative study. *Journal of Clinical Psychology*, **36**, 465–467.

Schulhoff, C. & Goodglass, H. (1969) Dichotic listening, side of brain injury and cerebral dominance. *Neuropsychologia*, **7**, 149–160.

Serafetinides, E. A. (1972) Laterality and voltage in the EEG of psychiatric patients. *Diseases of the Nervous System*, **33**, 622–623.

Siegenthaler, B. M. & Knellinger, L. D. (1981) Dichotic listening by brain-injured adults: observation of divergent test responses. *Communication Disorders*, **14**, 399–409.

Small, I. F. (1974) Inhalant convulsive therapy. In *Psychobiology of Convulsive Therapy* (eds M. Fink, S. Kety, J. McGaugh, *et al*). Washington, DC: V. H. Winston.

Spitzer, J. B. & Ventry, I. M. (1980) Central auditory dysfunction among chronic alcoholics. *Archives of Otolaryngology*, **106**, 224–229.

Squire, L. R. & Chace, P. M. (1975) Memory functions six to nine months after electroconvulsive therapy. *Archives of General Psychiatry*, **32**, 1557–1564.

——, —— & Slater, P. C. (1976) Retrograde amnesia following electroconvulsive therapy. *Nature*, **260**, 775–777.

——, Slater, P. C. & Miller, P. L. (1981) Retrograde amnesia and bilateral electroconvulsive therapy. *Archives of General Psychiatry*, **38**, 79–95.

——, Wetzel, C. D. & Slater, P. C. (1979) Memory complaint after electroconvulsive therapy: assessment with a new self-rating instrument. *Biological Psychiatry*, **14**, 791–801.

Steif, B. L., Sackheim, H. A., Portnoy, S., *et al* (1986) Effects of depression and ECT on anterograde memory. *Biological Psychiatry*, **21**, 921–930.

Templer, D. I., Ruff, C. T. & Armstrong, G. (1973) Cognitive functioning and degree of psychosis in schizophrenics given many electroconvulsive treatments. *British Journal of Psychiatry*, **123**, 441–443.

Wechsler, D. (1945) A standardized memory scale for clinical use. *Journal of Psychology*, **19**, 87–95.

—— (1981) *Manual for the Wechsler Adult Intelligence Scale – Revised*. New York: Psychological Corporation.

Weeks, D., Freeman, C. P. L. & Kendell, R. E. (1980) ECT: III: Enduring cognitive deficits? *British Journal of Psychiatry*, **137**, 26–37.

Wetherby, A. M., Koegel, R. L. & Mendel, M. (1981) Central auditory nervous system dysfunction in echolalic autistic individuals. *Speech & Hearing Research*, **24**, 420–429.

Yozawitz, A., Bruder, G., Sutton, S., *et al* (1979) Dichotic perception: evidence for right hemisphere dysfunction in affective psychosis. *British Journal of Psychiatry*, **135**, 224–237.

Zurif, E. B. & Ramier, A. M. (1972) Some effects of unilateral brain damage on the perception of dichotically presented phoneme sequences and digits. *Neuropsychologia*, **10**, 103–110

Karl M. Williams, PhD, *Department of Psychology, University of British Columbia, Vancouver, BC, Canada*; *William G. Iacono, PhD, *Department of Psychology, University of Minnesota, Minneapolis, MN 55455*; Ronald A. Remick, *Director of Affective Disorders Clinic, Department of Psychiatry, University of British Columbia, Vancouver, BC, Canada*; Patrick Greenwood, MA, *Audiology Clinic, Shaughnessy Hospital, Vancouver, BC, Canada*

*Correspondence

British Journal of Psychiatry (1990), **157**, 373–383

Towards a Cognitive-Behavioural Intervention for Adult Grief Reactions

D. J. KAVANAGH

Published accounts of behavioural interventions for grief have relied on exposure and habituation to grief cues as the primary strategy. Such an approach is excessively narrow, since it does not adequately confront the challenges that are posed by a bereavement. Many people cope with a bereavement by themselves, and for those, intervention may well be counterproductive. A cognitive-behavioural intervention, following models for depression/anxiety, can assist vulnerable individuals obtain a more rapid or complete adjustment.

There have been few published studies on behavioural treatment for pathological grief. Those that do exist focus on exposure to bereavement cues (Gauthier & Marshall, 1977; Gauthier & Pye, 1979; Mawson *et al*, 1981; Sireling *et al*, 1988). This approach is based on an analogy between grief and phobic reactions, in which stimuli that are associated with the bereavement take a similar status to phobic stimuli in provoking distress and avoidance (Ramsay, 1977). Exposure is thought to reduce the emotional response by habituation or counterconditioning. The outcome of this approach has been a substantial reduction in cue avoidance and associated distress, but differential effects on depressive mood have been less easy to replicate (Sireling *et al*, 1988).

In the study by Sireling *et al* (1988), a comparison was made between a group practising guided mourning, who were asked to confront the bereavement cues, and an anti-exposure group, who were instructed to avoid them. Both groups were given advice about relationships, work and leisure, which included encouragement to engage in new and enjoyable activities. The authors speculated that this advice was responsible for the similar rates of improvement in depressive mood for both groups. Without further research, this *post hoc* observation must be viewed cautiously, but the literature on outcome in depression suggests that the authors may be correct. Increasing pleasant activities is a powerful way of improving depressive mood (e.g. McLean & Hakstian, 1979; Zeiss *et al*, 1979; Wilson *et al*, 1983), and its inclusion in a treatment for intense or extended grief makes sense. Consistent with this view, people who quickly resume their usual social roles and activities have a more positive outcome from a bereavement than those who do not (Glick *et al*, 1974; Lund *et al*, 1985). While the observation does not establish a causal role for such activities, it contradicts the idea that such involvement necessarily implies avoidance and is countertherapeutic.

The results of Sireling *et al* (1988) suggest that behavioural approaches have suffered by being excessively bound to a phobic model of grief and not sufficiently applying an analogy with depression. If we give more consideration to the problems suffered by the bereaved and to the theoretical bases for treatment, we may be able to develop more successful approaches than those advances so far.

Evaluation of anxiety and depression models of grief

Aspects of grief do sometimes resemble an anxiety disorder, particularly early after the loss (Averill, 1968; Ramsay, 1977). The bereaved often experience physical anxiety symptoms or even panic attacks (Parkes, 1970). Many are plagued by distressing images of the deceased, by worries about their ability to cope with the future (Parkes & Brown, 1972) and by concerns over their own vulnerability (Krupnik & Horowitz, 1981). Anxiety-related cognitions can persist for long periods. Compared with married controls, widows report more worrying, more feelings of vulnerability and caution about close relationships for as long as four years after bereavement (Parkes & Brown, 1972).

However, the phobic model has difficulties with a number of other observations. Even in early bereavement, avoidance of stimuli is usually less prominent than a preoccupation with the deceased and a search for associated objects and environments (Parkes, 1970; Volkan, 1972). Similarly, 'delayed' or 'inhibited' grief (Parkes, 1965), which can be seen as a more prolonged and extreme form of avoidance, is only one type of complication of grief. The most common problem described by Parkes (1965) was 'chronic grief', where the person continues to be preoccupied with the loss and is significantly depressed for an extended period. The existence of chronic grief is a problem for a phobic model that relies on habituation or counterconditioning to explain recovery, because it represents an extensive series of exposure trials to multiple stimuli that has failed to produce a generalised reduction of distress. While this failure may be due to exposures being terminated before habituation can take place, there are no systematic data to support a rescue of the model on these grounds (cf. Parkes, 1965).

A more plausible explanation is that the depressive mood is being maintained by a separate process. This argument is supported by the absence of a differential impact on depression by the exposure-based treatment in Sireling *et al* (1988). Brasted & Callahan (1984) advanced a similar point of view when they characterised grief within behavioural paradigms of depression such as extinction and punishment, as well as in the phobic-like paradigm of aversive conditioning. A more cognitive perspective would characterise chronic grief in terms of an emotional feedback loop, where exposure to a sustained induction of deep sadness (such as a bereavement) triggers negative conditions, behaviour and physiological processes that serve to maintain and deepen the depressive mood (cf. Kavanagh, 1983; Kavanagh & Wilson, 1989; Teasdale, 1985).

A depression model receives support from a number of sources. Intense sadness is of course a usual effect of bereavement, as are other depressive features such as disturbances of sleep and appetite (Clayton *et al*, 1972). Recurrent bouts of sadness are commonly reported for a year or more after a bereavement (Parkes & Brown, 1972) and the risk of suicide increases significantly (Gove, 1972; MacMahon & Pugh, 1965). Even some data supportive of the anxiety model can be seen in terms of depression: what appears to be a quasiphobic avoidance of activities may often be better described as a depressive withdrawal, where the person no longer expects the activity to be pleasurable (Carson & Adams, 1980).

Application of a combined anxiety/depression model seems to be the best available clinical analogy, with grief appearing like a low-grade post-traumatic stress disorder. Such a model would incorporate most of the features that are observed in normal and chronic grief. It may also account for an increase in physical morbidity after a bereavement (Jacobs & Ostfeld, 1977; Stroebe & Stroebe, 1987), through a depressive neglect of health maintenance and perhaps through direct effects of sustained emotional reactions on cardiovascular or immune functioning (Jacobs & Ostfeld, 1977; Frederick, 1983; Pennebaker & Beall, 1986).

Survival of bereavement

Describing parallels between grief reactions and depression or anxiety does not mean that the reaction can usually be characterised as a major depressive disorder or anxiety disorder within DSM-III-R (American Psychiatric Association, 1987). Even after the death of a spouse, only about one-third develop a full depressive episode (Bornstein *et al*, 1973) and most people report some relief from dysphoria within three months (Parkes, 1970).

The relatively low correspondence between bereavement and major disorders has both theoretical and practical implications. In terms of practice, it suggests that entry into 'treatment' may give bereaved people the impression that we regard their difficulties as major disorders, and undermine their existing coping strategies. Theoretically, it raises the fascinating question of why major disorders are not more common after a bereavement. Here we have an event that generates powerful emotions and has long-lasting effects, yet most people seem to avoid a plunge into a major depressive episode.

Put in this way, bereavement is an extreme example of the typically low correlations between measures of life events and depression, where events *per se* account for only about 10% of the variance in depression (Brown & Harris, 1986). In the case of other events, commentators have often pointed to reliability and validity problems that are endemic in life-event instruments (Bebbington, 1986; Katschnig, 1986). For example, many rely on retrospective reports of events and some employ questionable methods for weighing events to form a total score (cf. Bebbington, 1986; Katschnig, 1986). But these considerations are less applicable to a bereavement whose occurrence is established, especially where the measure allows an assessment of the total environmental impact of the bereavement (Brown & Harris, 1986). Part of the explanation for the low rate of morbidity may be that instances of depression are being lost. It is true that the grief surveys are beset by refusal rates of around 40–50% (Maddison & Viola, 1968; Parkes & Brown, 1972; Clayton *et al*, 1972; Carey, 1977; Lund *et al*, 1985), and losses over the follow-up can reduce retention to as little as 26% of those who were originally contacted (Parkes & Brown, 1972). Despite some attempts to establish the representativeness of the final sample (e.g. Parkes & Brown, 1972), lost subjects may be more severely affected than participants (Vachon *et al*, 1982). Sometimes an assessment of emotional states is made retrospectively (e.g. Carey, 1977), allowing errors in the retrieval of negative reactions. Even in longitudinal studies of bereavement, episodes of depression may occur between assessments that are not being detected. However, it seems unlikely that inadequate measurement of events or disorders will provide a complete answer to the problem. I suggest that there are some important features of bereavement that provide a relatively benign outcome for most sufferers.

Grief is highly valued

In contrast to most instances of depressive mood, sadness immediately after the death of a close relative or friend usually meets with personal and social approval, and is viewed as a mark of emotional investment in the relationship. Empathic concern for the deceased is particularly valued as an altruistic and appropriate response. As a result, recently bereaved people can avoid the social opprobrium attached to psychiatric diagnosis and are less likely to experience secondary anxiety or depression about having a psychiatric disorder. A liberty to express negative emotion also allows the person to explore the implications of the death and habituate to distressing stimuli without having to be overly concerned about public control of their emotions. Of course, if the social reinforcement of grief reactions were indefinite, some survivors may be reluctant to readjust (Gauthier & Marshall, 1977). This problem is apparently averted by a combination of high incentives for adjustment and a withdrawal of social tolerance for the depression.

Stimulus exposure is hard to avoid

Usually there are many opportunities for exposure to stimuli that are associated with the bereavement. If the person was living in the same house as the deceased, daily routines provide constant reminders of the person. Similar effects are obtained from other places or activities that were associated with the deceased, and from attempts by other people to offer comfort. Aside from making radical changes, such as moving house, the only ways the bereaved can avoid this barrage of stimuli are to engage in a concerted programme of diverting attention or to hold onto the illusion that the deceased is still alive. These strategies are difficult to maintain in the face of frequent reminders that the deceased is absent.

There is often a continuation of other social roles

To the extent that the bereaved have occupational or child-rearing roles, there are activities they must continue to perform or must quickly resume after a brief respite. These activities provide a continuity of goals and daily schedules, and allow periods of distraction from the loss. Sometimes the activities may also offer moments of pleasure or success that help to moderate the depressive mood (Lewinsohn & Libet, 1972; Beck *et al*, 1979).

A bereavement can bring benefits

A bereavement can have substantial practical advantages for the survivor. A spouse has the freedom to seek a new partner and may have a new set of options for careers or leisure activities. Financially, some people are better off, and when the death occurred after a long illness, there can be legitimate relief over the end to the person's suffering (Shanfield *et al*, 1984). If survivors can avoid guilt over these sources of benefit, they can mitigate some of the effects of the loss and aid readjustment.

Some of the benefits from a bereavement arise in the course of meeting its challenges. In a study of parents who had lost a child from cancer two years before, Shanfield *et al* (1984) found that the majority saw themselves as closer to their remaining family and reported a sense of personal and spiritual growth. Most felt more able to express their feelings and discuss sensitive issues with others. As survivors begin to see increases in personal and social functioning as they adapted to the loss, these benefits can assist in their further adjustment.

There is an existing repertoire of coping skills

As Lazarus & Folkman (1984) observe, situational demands only become problematic when they exceed a person's capabilities. Most people approach a bereavement with a repertoire of coping skills that they have obtained from a variety of other unpleasant situations, from broken love affairs to failed examinations. Usually these situations will include at least one other bereavement. At least some of the existing coping skills will be applicable to the bereavement and moderate its aversive emotional impact. The more similar the bereavement is to previous stressors, the more applicable will be the skills that were acquired. Consistent with this perspective, Bornstein *et al* (1973) found a lower risk of depression among widows who had experienced a previous bereavement than for those who had not (4% v. 25%).

I suggest that generalised skills in controlling transient states of sadness fall into four main groups:

(a) skills in grappling with the situation and solving the problems that it poses (Goldfried & Goldfried, 1980)

(b) distraction and mood-induction skills, where attention may be directed towards positive thoughts and events or the person may increase the level of pleasant environmental stimulation (cf. Lewinsohn & Libet, 1972)
(c) skills in attacking cognitive sources of negative mood and countering its effects on incentives, self-efficacy and self-evaluation (Bandura, 1977; Beck *et al*, 1979; Schwartz & Clore, 1988)
(d) skills in mobilising assistance from other people, used to supplement deficiencies in any of the personal skills (Cohen & Wills, 1985).

These four sets of skills present a stability over time that may lead to their characterisation as personality factors, such as the 'hardness' concept of Kobasa *et al* (1982), but such a view implies an excessive resistance to improvement (Mischel, 1968). A skills approach is more optimistic about the potential for change.

Features of bereavement as a severely stressful event

The presence of generalised coping skills does not mean that they can be applied without modification to all stress situations. Bereavement presents a set of challenges that require a unique response.

Social norms restrict activities and the speed of adjustment

A rapid resumption of usual activities and mood is possible after an examination failure or even after the break-up of a relationship. In the case of grief, these changes may often violate norms about acceptable behaviour during mourning. This excludes the application of many of the generalised coping strategies that depend on environmental changes and induction of positive mood (set (b) of coping skills, above). It also can put bereaved people in a dilemma. A premature adjustment is unacceptable, but on the other hand a depressed mood has a number of disadvantages. It aids the retrieval of negative information from memory (Bower, 1981; Clark & Teasdale, 1982; Blaney, 1986; cf. Schwartz & Clore, 1990), it undermines confidence, and it reduces the power of incentives (Carson & Adams, 1980; Kanfer & Zeiss, 1983), so that coping is disrupted (Lewinsohn *et al*, 1980; Kavanagh, 1987). If the sad mood continues too long, supportive relationships are put under strain by self-absorption and a preference for negative topics of conversation (Gotlib & Robinson, 1982; Jacobsen & Anderson, 1982; Kuiper & McCabe, 1985). Bereaved people will run the risk of a full-blown depressive episode unless they control some of these reactions (Kavanagh, 1983; Kavanagh & Wilson, 1990; Teasdale, 1985).

Bereavement cues elicit extreme emotional responses

In common with other very severe events, a bereavement invests a large number of associated stimuli with the power to invoke intense emotional responses. This means that survivors will often need to ration their exposure to these cues if they are to control their mood, particularly early in bereavement. Seen in this light, avoidance of particularly evocative stimuli is an adaptive feature of early bereavement since it provides 'time out' from the distressing reality. On the other hand, some stimuli and memories of the deceased can blunt the completeness and permanence of the loss and this effect would be lost if the stimuli were avoided (Parkes, 1970; Volkan, 1972). For example, a coat may elicit vivid recollections of warm or humorous incidents involving the deceased, and the survivor may feel that contact with it forges an emotional link with the lost person.

Avoidance of too many stimuli also places a substantial constriction on thoughts and activities, so that it is difficult for the person to apply the coping skills in set (a) to solve the issues that are raised by the bereavement. Worse, the bereaved person remains vulnerable to powerful emotional responses when the stimuli are encountered. In this respect the problem is analogous to the one experienced by drug users who assiduously avoid stimuli that are associated with relapse, only to find that they are especially prone to relapse when they do encounter high risk situations (Cooney *et al*, 1983).

One tempting solution to the exposure problem in bereavement is to use alcohol or tranquillisers to dull the emotional response. Parkes & Brown (1972) found 28% of their sample reported increasing their alcohol intake since being widowed and 26% had started taking tranquillisers: the paper does not state the total number who had used chemical assistance. If it is used as a long-term solution the person is at risk of suffering physical and social damage (Babor *et al*, 1987). Sedation also reduces the person's cognitive resources to deal with the grief, and it does not build confidence in being able to face the cues alone. As a mood-control strategy the use of sedatives has limited application to bereavement.

Direct solution of the bereavement as a problem is difficult

The death of the deceased person is a permanent loss. While some of the practical and cognitive issues that attend the loss (such as financial problems or disruption of goals) are open to solution using set (a) of the coping skills, the deceased cannot be brought back except in fantasy or hallucination. Some people will be tempted to short-circuit the grieving process by quickly replacing the lost person with another. For example, parents sometimes react to the death of a child by quickly attempting to have another. This action has obvious value for species survival, but it may create additional pressures on mood control (such as increased emotionality during the pregnancy) and does not by itself represent an adequate solution to the loss (Condon, 1986).

Bereavement often presents a major challenge to central attitudes

A continuing problem for many people is the challenge that the death can pose to central attitudes by which we maintain goal-directed behaviour and challenge negative cognitions. Among these are beliefs about the meaningfulness and fairness of existence, and about our ability to control important events (Taylor, 1983; Abramson *et al*, 1978). Belief in a divine being that is totally good and loving towards humanity may also come under threat. Cognitive dissonance between the bereavement and these beliefs can add to feelings of anxiety. If the beliefs are relinquished, survivors may lose incentives for continued involvement in activities and perhaps for continuing to live at all. There is a temptation to resolve the dissonance by attributing the blame for the death to a specific person or institution. If this is someone else, the scapegoating induces anger, and if the anger continues it can disrupt social relationships and delay a recovery of positive mood. If bereaved people blame themselves, they engage in self-punitive thoughts and behaviour that from an objective standpoint are unnecessary and help to maintain a depressed mood. For example, bereaved parents may blame themselves for 'allowing' their adult children to travel on the route where a fatal accident occurred. A major focus of the cognitive skills that are required in a bereavement (set (c) above) is the resolution of the existential issues that it presents without falling prey to chronic anger, depressive nihilism, or negative self-attributions (Taylor, 1983).

Risk factors for severe reactions

Some deaths present more difficult challenges for a person's coping skills than do others. Another approach to understanding grief is to consider the risk factors for more severe or prolonged reactions.

Traumatic circumstances of the death

A bereavement through an accident or homicide frequently provokes a post-traumatic stress disorder that can last many years (Rynearson, 1984; Lehman *et al*, 1987). This may be especially severe if the person witnesses the death or if there is any implication that the survivor's actions contributed to the death (Martin *et al*, 1968; Nixon & Pearn, 1977). Suicide can be particularly problematic: not only are survivors often blamed for provoking it (Calhoun *et al*, 1986) but it can be seen as a rejection by the deceased. At the same time, it elicits intense empathy with the person's emotional suffering. Features that frequently accompany traumatic deaths include protracted legal investigations into the death that reinvoke negative cognition and emotion. If the body is not immediately recovered there is often considerable uncertainty about the manner or even the occurrence of death that creates additional difficulties for adjustment. In this situation survivors may also feel they lack a focus for mourning rituals and may believe that the deceased is not yet 'at peace'.

Another aspect of traumatic deaths is their unexpectedness, and some data suggest that prolonged illnesses may allow a faster adjustment than deaths after brief illnesses (e.g. Lundin, 1984). The rationale for the effect is that a long illness would provide opportunities for resolving past disagreements and allow the grieving process to begin before the death. However, the evidence for an expectedness effect is very weak (Clayton *et al*, 1973; Fulton & Gottesman, 1980; Sanders, 1982; Koocher, 1986). This means that if we add the period of distress during the illness to the period of adjustment after the death, a longer illness may actually prolong the total period of distress. Some of the special challenges presented by a long illness are reviewed by Koocher (1986) and Wortman & Dunkel-Schetter (1979).

Relationship with the deceased

As we might expect, emotionally close relationships provoke the most severe responses (Bugen, 1977), since they represent a greater loss of pleasure and have the potential to provoke stronger empathy for the deceased. Among family relationships, reactions to the death of spouses and children tend to be strongest (Fulton & Owen, 1977; Owen *et al*, 1982; Mor *et al*, 1986). Deaths of young people often elicit strong empathic responses about the brevity of their life and the limited range of their experience (Fulton & Owens, 1977). Parents may see their child's death as a failure to protect and care for the child (Nixon & Pearn, 1977).

Informal observations suggest that chronic grief by spouses is especially common after the termination of 'dependent' relationships, and that the risk is similar for partners who gave and for those who received support (Parkes, 1985). If so, the critical feature of dependent relationships may be their close reciprocal involvement. When a close relationship was also marked by significant disagreement or dissatisfaction, special difficulties for adjustment may be created by the confusion of emotional responses and the high potential for guilt and regret (Parkes & Weiss, 1983).

Presence of other problems

Other factors that increase the difficulty of a bereavement include unresolved issues from a previous bereavement (Volkan, 1972): the current event provides a potent retrieval cue for material that is associated with the past death, so that the person may have to deal with both losses at once. Difficulties with grief in the past may also be an index of the person's physiological or skill vulnerabilities in this situation. For similar reasons, a history of past psychiatric problems increases the risk of similar problems during a bereavement (Parkes, 1972; cf. Bornstein *et al*, 1973).

The death can also trigger a number of changes in life circumstances: for example, an elderly widow may move into an old people's home. Anxiety or depression over these circumstances may constitute an independent risk during bereavement (Brown & Harris, 1978; Helsing *et al*, 1981), although problems can also prove to be helpful diversions if they provide a source of activity and are easily resolved.

Absence of social support

Perhaps the most difficult feature of grief for many people is that the crisis is being faced without the assistance of the deceased, who was often a major source of social support. As in other crises, confiding relationships are a strong predictor of a positive outcome from the bereavement (Vachon *et al*, 1982; Pennebaker & O'Heeron, 1984; Cohen & Wills, 1985). In a study by Raphael (1977), perceived lack of support increased the risk of a poor health outcome at 13 months from under 20% to almost 86%. Confiding relationships can supplement a person's coping skills by providing a forum to discuss the turbulent cognitive-emotional reactions that are typically involved in grief, so that habituation of affect and informal cognitive therapy can take place.

Stages of grief

Some writers in the psychiatric literature (Engel, 1961; Kubler-Ross, 1970; Bowlby & Parkes, 1970) have advanced stage theories of grief that vary from a three-stage model by Engel (1961) to a five-stage theory by Kubler-Ross (1970). These models all begin with a stage of denial, emotional numbness, or shock. Then they have between one and three stages of awareness and disorganisation, and end with resolution or acceptance. The approach being advanced in the present paper is inconsistent with rigidly applied stages. Instead, it suggests that the nature and timing of grief reactions and coping will depend on the interaction between the challenges that an individual faces and the skills that he or she can bring to bear. The evidence seems to support this view. Emotional 'numbing' and a difficulty in acknowledging the death is most likely in the first month after the death, but only about half of bereaved people report experiencing it (Parkes, 1970). Resolution will by definition end a grieving process, but this does not make a significant contribution to our understanding of grief. Other stages are not universally experienced and do not occur in a predictable order, and reversion to previous stages is common (Bugen, 1977).

From the point of view of treatment, the danger in a naïve stage approach is the temptation to see early resolution of grief as a denial or postponement of grieving, and to encourage people to re-enter stages that we believe they have missed. In fact, a low level of emotional or somatic distress in the first two months is a predictor of good long-term adjustment (Vachon *et al*, 1982; Lund *et al*, 1985). Less than 2% of the subjects in the study by Vachon *et al* (1982) who had low distress scores soon after the bereavement had higher scores at later assessments. If we misconstrue signs of early improvement as evidence for avoidance, we may retard the person's adjustment to the loss.

Towards an intervention for grief

When we consider the complexity of grief and mourning, the inadequacy of a simple exposure model becomes painfully clear. Equally clear is that an exclusive emphasis on increasing pleasant events is inappropriate, since it will often clash with social norms and appear to lack empathy (Lehman *et al*, 1986). If applied in isolation it may also encourage an avoidance of the substantive issues that are posed by the death. That is not the route that I suggest. Rather, I propose that a cognitive-behavioural intervention for grief should be an individualised approach that is based on a functional analysis and takes into account each of the issues raised in the foregoing discussion. The intervention will usually represent a compromise between aspects that encourage exploration of the loss and ones that moderate the depressive response. Its features include items under the following headings.

Recognition of the value placed on grief and mourning

The intervention recognises the benefits obtained from conformity to personal and social rules about mourning, and is preceded by a careful assessment of these norms (Eisenbruch (1984*a*,*b*) and Rosenblatt *et al* (1976) examine cultural variations in mourning). Before beginning to apply a new strategy that may violate these expectations (such as increasing social activity), normative issues are discussed and irrational guilt is minimised. A decision to proceed is made by the survivor after considering the potential costs and benefits of alternative actions.

Recognition of existing skills

For many bereaved people, entry into 'therapy', however well meaning, will be counterproductive. They bear their pain with dignity and assurance, they avoid irrational anger and guilt, and they have relationships that help them to bear the loss. To say that such people need assistance for a disorder of grief is to ask for an admission of defeat and invites the opprobrium attached to psychiatric diagnoses. For this reason an intervention is primarily for people who seek treatment and for the high-risk groups outlined above (Raphael, 1977). It is presented within an educational framework that builds on existing skills to cope with stress and encourages self-management. Where possible, aspects may be conducted through self-help groups (Vachon *et al*, 1980), which have the dual advantages of greater acceptability and the presence of credible models (Bandura, 1986).

Controlled exposure to bereavement cues

As in the 'guided-mourning' approach (Mawson *et al*, 1981; Sireling *et al*, 1988), survivors are encouraged to confront environmental and cognitive stimuli that are associated with the bereavement. This aspect of the intervention will be especially important when the survivor is avoiding bereavement cues or is having a post-traumatic reaction to the death. Encounters with bereavement stimuli are not simply seen in a counterconditioning or habituation paradigm, but involve the development of self-efficacy and skills to face the stimuli alone (Bandura, 1977, 1986; set (a) of the stress coping skills above). Among these skills are attention training, where the person focuses on the target cue and temporarily ignores other emotive stimuli. This training may not only improve the effectiveness of exposure trials between sessions, but can be used when rehearsing positive memories of the deceased that are perceived as valuable or comforting. Encounters with bereavement stimuli also provide an opportunity to grapple with some of the practical and philosophical issues that it raises. Accordingly, they are used to build skills in defining problems, gathering evidence, developing alternatives, and reviewing options to resolve the issue (cf. Beck *et al*, 1979; Goldfried & Goldfried, 1980).

Exposure need not be unremitting and constant for therapeutic benefit. As long as survivors are obtaining practice in encounters with each of the important cues, they may avoid them on occasions when relief from emotional distress is required. For this reason the method is termed 'controlled exposure', in which survivors become empowered both to confront the stimuli and to control the extent of depressive ruminations. A distinction is drawn between cognitive-emotional reactions that are elicited automatically and deliberate attention to bereavement cognitions. To control automatic responses, the person identifies the situations where there is a high risk of distressing thoughts, and plans participation with this risk in mind (cf. Marlatt & Gordon, 1985). Attention skills are used to focus on positive stimuli between exposure trials and deliberate consideration of cognitive issues is limited to a systematic analysis at restricted times. Control of distressing stimuli is presented as 'time out' from the tasks of bereavement that will help maintain the strength to face them squarely.

Graduated involvement roles and activities

An increase in the level and range of activities has several purposes. When it allows a temporary distraction from the loss and gives moments of pleasure it helps survivors to control the depth of their sadness (Lewinsohn & Libet, 1972). It also provides a test of negative cognitions through recording mastery and pleasure (Beck *et al*, 1979). Activities to fulfil these aims are selected by their relevance to problematic cognitions and their potential for pleasure or success.

Sometimes re-involvement will also involve some confrontation of grief rather than a distraction, especially if the activities were previously undertaken together. If so, any intrinsic pleasure from the activity may initially be overshadowed by sadness and the immediate emotional benefit will come from a sense of achievement in confronting the bereavement associations. Activities such as these may also be difficult because survivors are preoccupied with bereavement thoughts and are unable to muster sufficient attention to deal with the task at hand. In this case, the objective is to develop attentional strategies to maximise the achievement. Sometimes the task's challenge derives from other impairments that are triggered by a sad mood. For example, social activities are often very difficult when a person is feeling depressed, in part because the sad mood interferes with voice tone, smiling and appreciation of humour. At first, the social activities will need to be consistent with these performance features and might include contacts with people who have recently recovered from a bereavement or dinner with close friends: attending a party or going on a date would probably come much later. Within sessions, the person practises performing the skills that will be required for forthcoming activities.

Before a person is willing to engage in an activity, it will often be necessary to boost the incentives for attempting it. Not only are incentives reduced by the depressive mood (Carson & Adams, 1980), but they often involve the deceased either as the source of rewards or the recipient of benefits. For example, cooking a special meal may have been maintained by expected approval from the deceased or by the expected pleasure he or she would obtain. These incentives will need to be replaced by others such as self-reward for the achievement or temporary reward by the therapist if the person is to undertake the activity. Incentives may sometimes be easier to establish with new activities that bear no association with the deceased, although such activities will almost certainly require a set of new skills to ensure a successful performance. The process of forming new goals and self-regulatory processes will need special attention when the survivor and deceased had a closely meshed relationship such as the 'dependent' type discussed by Parkes (1985). When these relationships dissolve, there may be little motivation for basic everyday activities, and the development of goals and incentives may prove critical for prevention of suicide.

Cognitive therapy for excessively negative conditions

Cognitive therapy, after Beck *et al* (1979), is part of controlled exposure and renewed involvement, but its importance demands separate mention. Survivors are assisted in identifying specific thoughts that are associated with their most severe emotional responses. In a Socratic style, the therapist asks them to seek out the evidence for especially negative cognitions and examine their validity. The objective is to control aversive moods by replacing overly negative thoughts with more positive and functional ones. When applying cognitive therapy to grief, particular attention is paid to hopelessness about the future and to cognitions of guilt or personal worthlessness. A focus on guilt is especially important when survivors are being blamed for the death or when there were problems in their relationship with the deceased. If there is a rational basis for guilt, survivors are asked to determine the extent and duration of self-punishment that is reasonable and the ways that are available for symbolic restitution.

A similar approach is taken to anger. Within a social-cognitive model anger is not seen as an emotion that requires cathartic expression before it is dissipated (Bandura, 1986). Instead, chronic anger is viewed as a series of mood inductions that achieve persistence from their bases in repetitive cognitions about aversive experiences and their unfairness. This model is presented to survivors, who then examine the validity of their triggering cognitions and the functional value of their expressions of anger. Irrational aspects are substituted by more adaptive thoughts which the person rehearses when the cognitions occur. Where possible, the survivor generates strategies to resolve the issues at the root of the anger (e.g. by inquiries about the cause of death).

Maximising the availability of social support

Survivors are asked to review potential sources of practical and emotional support among their existing social network. They are encouraged to see assistance from others as a legitimate temporary measure to allow them to weather crises and develop skills and resources to continue on their own. To ensure that continued support will be available, they attempt selected tasks by themselves and diversify their sources of support as much as possible. From the beginning they try to maintain empathy with those who give support, and find ways to preserve the mutual pleasure of encounters with them.

Control of drug and alcohol use

Sometimes the use of alcohol or other drugs can retard a survivor's progress in acquiring coping skills and meeting the challenges that are posed by the bereavement. Drugs can also produce secondary problems in health or social functioning. Unless physical dependence is acute, a self-control strategy is applied to limit the drug intake to appropriate levels (Miller & Munoz, 1982; Heather & Robertson, 1983). At the same time, survivors are taught alternative strategies to control their distress and increase their sleep, such as progressive muscular relaxation (Bernstein & Borkovec, 1973) and other cognitive-behavioural techniques (Coates & Thoreson, 1977).

Comparison with previous interventions

Interventions like the one described above have been used by practitioners for some time, and have been discussed by writers in the dynamic tradition such as Raphael (1975) and Worden (1983). The present approach differs from dynamic treatments by placing less emphasis on defensive behaviour, insight and interpretation, and more on training of coping skills. However, in practice the dynamic treatments may closely match the strategies suggested here.

The present intervention departs from the 'guided-mourning' approach in the study by Sireling *et al* (1988) by legitimising controlled distraction and explicitly including cognitive therapy and skills training. Resumption of activity is now seen as a central feature of the antidepressive strategy and social support from other sources is fostered. As a result, the intervention encompasses a greater variety of grief problems that are encountered in clinical practice, and formally recognises what many cognitive-behavioural practitioners are actually doing. In promoting flexibility and an ideographic approach to treatment, it also accords more closely with the spirit of the behavioural tradition.

Surviving grief as a positive achievement

Therapists tend to focus on the negative outcomes of grief, and some writers have even suggested that we should see grief as an illness (e.g. Engel, 1961). In contrast, the present paper has focused on bereavement as a challenge, and on the adaptive capabilities that most people have to meet it. Their successful adjustment to it is not just an absence of disorder, but represents a positive achievement, with corresponding increases in skills. The overall aim of the present intervention is to maximise survivors' achievements and minimise the pain they suffer to gain them.

Acknowledgements

This paper was completed while the author was on study leave at the MRC Social Psychiatry Unit, Institute of Psychiatry, London. Comments by Paul Bebbington, Fiona Blyth, Dominic Lam, Michael Nicholas and Nick Tarrier on an earlier draft are gratefully acknowledged.

References

Abramson, L. Y., Seligman, M. E. P. & Teasdale, J. D. (1978) Learned helplessness in humans: critique and reformulation. *Journal of Abnormal Psychology*, **87**, 49–74.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

Averill, J. R. (1968) Grief. its nature and significance. *Psychological Bulletin*, **70**, 721–748.

Babor, T. F., Kranzler, H. R. & Lauerman, R. J. (1987) Social drinking as a health and psychosocial risk factor: Anstie's limit revisited. In *Recent Developments in Alcoholism, vol. 5* (ed. M. Galanter). New York: Plenum.

Bandura, A. (1977) Self-efficacy: toward a unifying theory of behavioural change. *Psychological Review*, **84**, 191–215

—— (1986) *Social Foundations of Thought and Action*. Englewood Cliffs, NJ: Prentice-Hall.

Bebbington, P. (1986) Establishing causal links – recent controversies in *Life Events and Psychiatric Disorders: Controversial Issues* (ed. H. Katschnig), pp. 188–200. Cambridge: Cambridge University Press.

Beck, A. T., Rush, A. J., Shaw, B. F., *et al* (1979) *Cognitive Therapy of Depression*. New York: Wiley.

Blaney, P. H. (1981) The effectiveness of cognitive and behavioral therapies. In *Behavior Therapy for Depression: Present Status and Future Directions* (ed. L. P. Rehm). New York: Academic Press.

—— (1986) Affect and memory: a review. *Psychological Bulletin*, **99**, 229–246.

Bernstein, D. A. & Borkovec, T. D. (1973) *Progressive Muscular Relaxation Training*. Champaign, Ill: Research Press.

Bornstein, P. E., Clayton, P. J., Halikas, J. A., *et al* (1973) The depression of widowhood after thirteen months. *British Journal of Psychiatry*, **122**, 561–566.

Bowlby, J. & Parkes, C. M. (1970) Separation and loss. In *The Child and his Family: vol. 1. The International Yearbook of Child Psychiatry and Allied Professions* (eds E. J. Anthony & C. Koupernik), pp. 197–216. New York: Wiley.

Bower, G. (1981) Mood and memory. *American Psychologist*, **36**, 129–148.

Brasted, W. S. & Callahan, E. J. (1984) A behavioral analysis of the grief process. *Behavior Therapy*, **15**, 529–543

Brown, G. W. & Harris, T. O. (1978) *Social Origins of Depression: A Study of Psychiatric Disorder in Women*. London: Tavistock.

—— & —— (1986) Establishing causal links: the Bedford College studies of depression. In *Life Events and Psychiatric Disorders: Controversial Issues* (ed. H. Katschnig), pp. 107–187. Cambridge: Cambridge University Press.

Bugen, L. A. (1977) Human grief: a model for prediction and intervention. *American Journal of Orthopsychiatry*, **47**, 196–206.

Calhoun, L. G., Selby, J. W. & Walton, P. B. (1986) Suicidal death of a spouse: the social perception of the survivor. *Omega*, **16**, 283–288.

Carey, R. G. (1977) The widowed: a year later. *Journal of Consulting Psychology*, **24**, 125–131.

Carson, T. P. & Adams, H. E. (1980) Activity valence as a function of mood change. *Journal of Abnormal Psychology*, **89**, 368–377.

Clark, D. M. & Teasdale, J. D. (1982) Diurnal variation in clinical depression and accessibility of positive and negative experiences. *Journal of Abnormal Psychology*, **91**, 87–95.

Clayton, P. J., Halikas, A. & Maurice, W. L. (1972) The depression of widowhood. *British Journal of Psychiatry*, **120**, 71–77.

——, ——, ——, *et al* (1973) Anticipatory grief and widowhood. *British Journal of Psychiatry*, **122**, 47–51.

Coates, T. J. & Thoreson, C. E. (1977) *How to Sleep Better: A Drug Free Program for Overcoming Insomnia*. Englewood Cliffs, NJ: Prentice-Hall.

Cohen, S. & Wills, T. A. (1985) Stress, social support and the buffering hypothesis. *Psychological Bulletin*, **98**, 310–357.

Condon, J. T. (1986) Management of established pathological grief reaction after stillbirth. *American Journal of Psychiatry*, **143**, 987–992.

Cooney, N. L., Baker, L. & Pomerleau, O. F. (1983) Cue exposure for relapse prevention in alcohol treatment. In *Advances in Clinical Behavior Therapy* (eds R. J McMahon & K. D. Craig). New York: Brunner/Mazel.

Eisenbruch, M. (1984*a*) Cross-cultural aspects of bereavement. I: A conceptual framework for comparative analysis. *Culture, Medicine, and Psychiatry*, **8**, 283–309.

—— (1984*b*) Cross-cultural aspects of bereavement. II: Ethnic and cultural variations in the development of bereavement practices.

Engel, G. L. (1961) Is grief a disease? *Psychosomctic Medicine*, **23**, 18–22.

Frederick, J. F. (1983) The biochemistry of bereavement: possible basis for chemotherapy? *Omega*, **13**, 295.

Fulton, R. & Owen, G. (1977) *Adjustment to Loss Through Death: A Sociological Analysis*. University of Minnesota: Center for Death Education and Research.

—— & Gottesman, D. J. (1980) Anticipatory grief: a psychosocial concept revisited. *British Journal of Psychiatry*, **137**, 45–54.

Gauthier, J. & Marshall, W. L. (1977) Grief: a cognitive-behavioral analysis. *Cognitive Therapy and Research*, **1**, 39–44.

—— & Pye, C. (1979) Graduated self-exposure in the management of grief. *Behaviour Analysis and Modification*, **3**, 202–208.

Glick, I. O., Weiss, R. S. & Parkes, C. M. (1974) *The First Year of Bereavement*. New York: John Wiley.

Goldfried, M. R. & Goldfried, A. P. (1980) Cognitive change methods. In *Helping People Change* (eds F. H. Kanfer & A P. Goldstein). New York: Pergamon.

Gotlib, I. H. & Robinson, L. A. (1982) Responses to depressed individuals: discrepancies between self-report and observer-rated behavior. *Journal of Abnormal Psychology*, **91**, 231–240.

Gove, W. R. (1972) Sex, marital status, and suicide. *Journal of Health and Social Behavior*, **13**, 204–213.

Heather, N. & Robertson, I. (1983) *Controlled Drinking*. London: Heath.

Helsing, K. J., Szklo, M. & Comstock, G. W. (1981) Factors associated with mortality after widowhood. *American Journal of Public Health*, **71**, 802–809.

Jacobs, S. & Ostfeld, A. (1977) An epidemiological review of the mortality of bereavement. *Psychosomatic Medicine*, **39**, 344–357.

Jacobsen, N. S. & Anderson, E. A. (1982) Interpersonal skill and depression in college students: an analysis of the timing of self-disclosures. *Behavior Therapy*, **13**, 271–282.

Kanfer, R. & Zeiss, A. M. (1983) Depression, interpersonal standard-setting, and judgments of self-efficacy. *Journal of Abnormal Psychology*, **92**, 319–329.

Katschnig, H. (1986) Measuring life stress – a comparison of the checklist and the panel technique. In *Life Events and Psychiatric Disorders: Controversial Issues* (ed. H. Katschnig), pp. 74–106. Cambridge: Cambridge University Press.

Kavanagh, D. J. (1983) Influence of joy and sadness on efficacy and task selection. (Doctoral dissertation, Stanford University.) *Dissertation Abstracts International*, **44**, 2922B.

—— (1987) Mood, persistence, and success. *Australian Journal of Psychology*, **39**, 307–318.

—— & Wilson, P. H. (1989) Prediction of outcome with a group version of cognitive therapy for depression. *Behaviour Research and Therapy*, **27**, 333.

Kobasa, S. C., Maddi, S. R. & Kahn, S. (1982) Hardiness and health: a prospective study. *Journal of Personality and Social Psychology*, **42**, 168–171.

Koocher, G. P. (1986) Coping with a death from cancer. *Journal of Consulting and Clinical Psychology*, **54**, 623–631.

Krupnik, J. L. & Horowitz, M. J. (1981) Stress response syndromes: recurrent themes. *Archives of General Psychiatry*, **38**, 428–435.

Kubler-Ross, E. (1970) *On Death and Dying*. London: Tavistock.

Kuiper, N. A. & McCabe, S. B. (1985) The appropriateness of social topics: effects of depression and cognitive vulnerability on self and other judgments. *Cognitive Therapy and Research*, **9**, 371–379.

Lazarus, R. S. & Folkman, S. (1984) *Stress, Appraisal and Coping*. New York: Springer

Lehman, D. R., Ellard, J. & Wortman, C. B. (1986) Social support for the bereaved: recipients' and providers' perspectives on what is helpful. *Journal of Consulting and Clinical Psychology*, **54**, 438–446.

——, Wortman, C. R. & Williams, A. F. (1987) Long-term effects of losing a spouse or child in a motor vehicle crash. *Journal of Personality and Social Psychology*, **52**, 218–231.

Lewinsohn, P. & Libet, J. (1972) Pleasant events, activity schedules, and mood. *Journal of Abnormal Psychology*, **79**, 291–295.

——, Mischel, W., Chaplin, W., *et al* (1980) Social competence and depression: the role of illusory self-perceptions. *Journal of Abnormal Psychology*, **89**, 194–202.

Lund, D. A., Dimond, M. F., Caserta, M. S., *et al* (1985) Identifying elderly with coping difficulties after two years of bereavement. *Omega*, **16**, 213–224.

Lundin, T. (1984) Long-term outcome of bereavement. *British Journal of Psychiatry*, **145**, 424–428.

MacMahon, B. & Pugh, T. F. (1965) Suicide in the widowed. *American Journal of Epidemiology*, **81**, 23–31.

Maddison, D. & Walker, W. L. (1967) Factors affecting the outcome of conjugal bereavement. *British Journal of Psychiatry*, **113**, 1057–1067.

—— & Viola, A. (1968) The health of widows in the year following bereavement. *Journal of Psychosomatic Research*, **12**, 297–306.

Marlatt, A. & Gordon, J. R. (1985) *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviours*. New York: Guildford.

Martin, H. L., Lawries, J. H. & Wilkinson, A. W. (1968) The family of the fatally burned child. *Lancet*, *i*, 628–629.

Mawson, D., Marks, I. M., Ramm, L., *et al* (1981) Guided mourning for morbid grief: a controlled study. *British Journal of Psychiatry*, **138**, 185–193.

McLean, P. D. & Hakstian, A. R. (1979) Clinical depression: comparative efficacy of outpatient treatments. *Journal of Consulting and Clinical Psychology*, **47**, 818–836.

Miller, W. R. & Munoz, R. F. (1982) *How to Control Your Drinking*. Albuquerque, New Mexico: New Mexico Press.

Mischel, W. (1968) *Personality and Assessment*. New York: Wiley.

Mor, V., McHorney, C. & Sherwood, S. (1986) Secondary morbidity among the recently bereaved. *American Journal of Psychiatry*, **143**, 158–163.

Nixon, J. & Pearn, J. (1977) Emotional sequelae of parents and siblings following the drowning or near-drowning of a child. *Australian and New Zealand Journal of Psychiatry*, **11**, 265–268.

Owen, G., Fulton, R. & Markusen, E. (1982) Death at a distance: a study of family survivors. *Omega*, **13**, 191.

Parkes, C. M. (1965) Bereavement and mental illness. Part 2. A classification of bereavement reactions. *British Journal of Medical Psychology*, **38**, 13–26.

—— (1970) The first year of bereavement: a longitudinal study of the reaction of London widows to the death of their husbands. *Psychiatry*, **33**, 444–467.

—— (1972) *Bereavement. Studies of Grief in Adult Life*. London: Tavistock.

—— (1985) Bereavement. *British Journal of Psychiatry*, **146**, 11–17.

—— & Brown, R. J. (1972) Health after bereavement: a controlled study of young Boston widows and widowers. *Psychosomatic Medicine*, **34**, 449–461.

—— & Weiss, R. S. (1983) *Recovery from Bereavement*. New York: Basic Books.

Pennebaker, J. W. & O'Heeron, R. C. (1984) Confiding in others and illness rate among spouses of suicide and accidental death victims. *Journal of Abnormal Psychology*, **93**, 473–476.

—— & Beall, S. K. (1986) Confronting a traumatic event: toward an understanding of inhibition and disease. *Journal of Abnormal Psychology*, **95**, 274–281.

Ramsay, R. W. (1977) Behavioural approaches to bereavement. *Behavour Research and Therapy*, **15**, 131–135.

Raphael, B (1975) The management of pathological grief. *Australian and New Zealand Journal of Psychiatry*, **34**, 1450–1454.

—— (1977) Preventative intervention with the recently bereaved. *Archives of General Psychiatry*, **34**, 1450–1454.

Rosenblatt, P. C., Walsh, R. P. & Jackson, S. A. (1976) *Grief and Mourning in Cross-cultural Perspective*. New Haven: HRAF Press.

Rynearson, E. K. (1984) Bereavement after homicide: a descriptive study. *American Journal of Psychiatry*, **141**, 1452–1454.

Sanders, C. M. (1982) Effects of sudden vs. chronic illness death on bereavement outcome. *Omega*, **13**, 227–241.

Schwartz, N. & Clore, G. L. (1990) How do I feel about it? The informative function of affective states. In *Affect, Cognition, and Social Behavior* (ed. K. Fielder & J. Forgas), p. 44. Toronto: Hogrefe International.

Shanfield, S. B., Benjamin, A. H. & Swain, B. J (1984) Parents' reactions to the death of an adult child from cancer. *American Journal of Psychiatry*, **149**, 1092–1094.

Sireling, L., Cohen, D. & Marks, I. (1988) Guided mourning for morbid grief: a replication. *Behavior Therapy*, **19**, 121–132.

Stroebe, W. & Stroebe, M S. (1987) *Bereavement and Health: The Psychological and Physical Consequences of Partner Loss*. Cambridge: Cambridge University Press.

Taylor, S. (1983) Adjustment to threatening events: a theory of cognitive adaptation. *American Psychologist*, **38**, 1161–1173.

Teasdale, J. D (1985) Psychological treatments for depression: how do they work? *Behaviour Research and Therapy*, **23**, 157–165.

Vachon, M. L. S., Lyall, W. A. L., Lancee, W. J., *et al* (1980) A controlled study of a self-help intervention for widows. *American Journal of Psychiatry*, **137**, 1380–1384.

——, Rogers, J., Lyall, A., *et al* (1982) Predictors and correlates of adaptation to conjugal bereavement. *American Journal of Psychiatry*, **139**, 998–1002.

Volkan, V. (1972) The linking objects of pathological mourners. *Archives of General Psychiatry*, **27**, 215–221.

Wilson, P. H., Goldin, J. C. & Charbonneau-Powis, M. (1983) Comparative efficacy of behavioral and cognitive treatments for depression. *Cognitive Therapy and Research*, **7**, 111–124

Worden, J. W. (1983) *Grief Counselling and Grief Therapy*. London: Tavistock.

Wortman, C. B. & Dunkel-Schetter, C. (1979) Interpersonal relationships and cancer: a theoretical analysis *Journal of Social Issues*, **35**, 120–155.

Zeiss, A. M., Lewinsohn, P. M. & Munoz, R. F. (1979) Non-specific improvement effects in depression using interpersonal skills training, pleasant activity schedules, or cognitive training. *Journal of Consulting and Clinical Psychology*, **47**, 427–439.

David J. Kavanagh BA, PhD, *Lecturer in Psychology, Department of Psychology*, Badham Clinic, Building A16, University of Sydney 2006, Australia

British Journal of Psychiatry (1990), 157, 384–388

The Value of the Claybury Selection Battery in Predicting Benefit from Group Psychotherapy

MARK J. PEARSON and ALAN J. GIRLING

Before receiving psychodynamic group psychotherapy at the Uffculme Clinic four groups of out-patients were assessed on the Claybury Selection Battery and the SCL 90, and therapists were asked to predict their progress. Outcome was assessed at about three months and one year by patients' self-reports of symptom change on the SCL 90 and therapists' ratings of progress. Only one of the three Claybury questionnaires, the TEQ, which measures attitudes to treatment, was significantly correlated with outcome, along with therapists' predictions. This result is different to that of a previous study which also found that outcome correlated with direction of interest (DIQ) and conservatism (C). The C and DIQ scales may provide useful predictive information only in a small proportion of cases.

The problem of predicting which patients are likely to benefit from psychodynamic and behavioural approaches to psychotherapy was addressed by Caine *et al* (1981) at the Claybury Hospital in Essex. Taking a view put forward by Kelly (1955) and Goldstein (1962) they argued that psychotherapy is more likely to be effective when there is a good fit between the personality, values and expectations of the patient and the values implicit in the therapy. Thus, small group psychodynamic psychotherapy, which focuses on exploring feelings and personal relationships, is likely to appeal to people who prefer to turn their attention to matters of the imagination ('inner-directed'), who have liberal rather than conservative attitudes, and who feel that therapy should involve sharing problems with fellow patients rather than receiving advice from an expert. The result of their work was The Claybury Selection Battery (Caine *et al*, 1982), which includes three patient self-report questionnaires designed to measure direction of interest (the DIQ), conservatism (the 'C' scale), and treatment expectancies (the TEQ).

This study tested one half of the Claybury workers' predictions, namely that there will be a positive correlation between good outcome in psychodynamic group therapy and specific pre-therapy scores on the Claybury questionnaires (high DIQ, low TEQ, low C). It is comparable with the Caine & Wijesinghe (1976) study, in that the patients, therapists, and therapy are all broadly similar.

The two studies differ in their methods of assessing outcome, however. Caine & Wijesinghe (1976) began with a sample of 100 patients and used two methods. First, they asked therapists to rate response to therapy after a nine-month period: 18 patients were rated as responding well, eight were still in therapy but rated as not responding, and 52 patients had already dropped out. The latter two groups were combined to form a group of 60 non-responders, and it was found that these patients had significantly lower DIQ scores and higher TEQ scores than the 18 responders. The main problem with this method is that it rests on the assumption that premature termination of therapy indicates non-response. The second method used was to send a postal questionnaire to patients two years after the end of therapy. Only 24 patients responded and, of these, 12 felt that they had benefited from group therapy and 12 did not. The pre-test scores of these two groups showed significant differences on all three Claybury questionnaires, along the lines predicted, but the numbers in each group were now small and represented only 24% of the initial sample.

Given these methodological problems, a replication of the work was carried out. Outcome was assessed by patients' ratings of symptom improvement and therapists' ratings of progress within the group. These assessments were made at three months and one year and might be called measures of progress rather than final outcome, since most groups at the Uffculme Clinic are slow, open groups in which patients can stay for more than one year. However, the theory behind the Claybury questionnaires (Caine *et al*, 1981) does seem to argue that the effects of matching between personal styles and therapy characteristics will be detectable from the outset. Indeed, Caine & Wijesinghe (1976) reported significant correlations at nine months.

In addition, this study compared the predictive power of the Claybury questionnaires with the normal clinical method of predicting progress in group therapy, namely therapists' predictions. The value of the Claybury Battery is increased if it can provide information which is not already available to therapists in the form of their own clinical predictions. With this in mind, it was decided to perform an item analysis of any of the Claybury questionnaires which showed a significant correlation with the outcome measures in an attempt to understand why the questionnaire was successful.

Method

The research was conducted between November 1985 and April 1987 at the Uffculme Clinic, the regional centre for psychotherapy in the West Midlands. All experienced therapists setting up new groups were approached and four groups were recruited, each consisting of eight patients and two therapists. Therapists followed their normal procedure for the selection and composition of their groups. In all cases this included an assessment interview with a psychiatrist and the provision of a standard ten-page handout developed by the clinic to introduce patients to the principles of psychodynamic group therapy. Patients with psychotic symptoms were excluded at the assessment stage.

The predictive tests (Claybury questionnaires, and therapists' predictions) were completed at assessment or after the first group session. At the same time, patients were asked to rate the severity of 90 common symptoms over the preceding four-week period using the Symptom Checklist 90 (SCL 90; Derogatis, 1981). At three months and one year, patients repeated the SCL 90 and therapists rated their progress in group to date. Finally the TEQ questionnaire was repeated by all patients who remained in therapy at one year to see if their treatment expectancies had changed. Patients were asked to make their SCL 90 ratings at home in the week between group sessions so that their ratings were not biased by following an emotionally charged experience.

Therapists' ratings were made on a predictions and outcome form specially designed for the project (copies available from author). The form asks therapists to place patients in one of three categories as follows: 'A' is good outcome where the patient is involved with the group and able to use the group to make progress with the presenting problems; 'B' is average outcome where the patient is involved some of the time, but has doubts about the usefulness of therapy, may have missed occasional sessions and made only small improvements in symptoms and patterns of relationships within the group; and 'C' is poor outcome where the patient remains peripheral and uninvolved or has terminated with no progress. Each therapist was asked to make their assessment independently while blind to the ratings of their co-therapist and the Claybury and SCL 90 data. For the purposes of statistical analysis, the categorical ratings of the co-therapists were combined to give a numerical score using the system: AA = 1, AB = 2, BB = 3, BC = 4, CC = 5. To allow for further discrimination, therapists were asked to rank patients placed within the same category. This produces two types of therapists' scores, one based on categories with a range (1–5) that indicates therapists' assessment of the quality of progress, and one based on rankings with a wider range (2–16) which solely represents the progress of patients relative to each other.

Attempts were made to assess the outcome of all the 32 patients who started in therapy, including those who had left the group early. Leavers were assessed by therapists using the same form but basing their ratings on the progress made up until the time of leaving. Leavers were sent the SCL 90 questionnaire through the post at the two follow-up points, but in practice only one returned the form, with the result that only 30 patients could be assessed in terms of symptoms at three months and only 22 at one year.

There was good agreement between the two therapists involved in assessing each group. Concordance between the rankings was measured by Kendall's coefficient (equivalent to the ordinary rank correlation coefficient when just two assessors rank each case). The average coefficient was 0.75 for the predicted ratings and 0.86 at the outcome stages. The categorical ratings from the two assessors gave different results in just 22% of cases at the prediction stage and 14% of cases at the outcome stages. In no instance was a patient given an 'A' rating by one assessor and a 'C' rating by the other.

Patients

Demographic information on the 32 patients was collected from the case notes. There were 20 women and 12 men, and the mean age of the sample was 33 years (range 19–50). A total of 21 were in full time employment, five were unemployed, five were housewives and there was one full-time student. The main psychiatric diagnosis at assessment was a neurotic disorder in 26 cases, and a personality disorder in six. The mean score on the Global Severity Index (GSI) of the SCL 90 at intake was 1.65 (s.d. 0.57) which indicates that this patient group rated themselves as having significantly more symptoms ($P < 0.01$) than that described by Derogatis *et al* (1976), where the mean score was 1.30 (s.d. 0.59). The mean Claybury scores at intake were: DIQ = 14.5 (s.d. 6.0), TEQ = 30.7 (s.d. 6.8), C scale = 43.7 (s.d. 10.0).

Therapists

Six of the eight therapists would be considered experienced in running psychodynamic groups in that they were working full time within the clinic and were involved in groups of this type on a daily basis under supervision. One therapist was a consultant psychotherapist, four were psychiatrists in training, two were registered mental health nurses working full time in psychotherapy and one was a teacher with a special interest in psychotherapy. None were psychoanalysts.

Groups

Three of the four groups were mixed-sex groups with equal numbers of men and women, led by mixed co-therapy teams. In the fourth group, all the patients and the two therapists were women. The different composition of this group arose because there were a number of women patients who had expressed a preference for an all female group. However, the therapists in this group had both run

TABLE I
Numbers of patients continuing in therapy at three months and one year and therapists' ratings of their progress

Co-therapists' ratings[1]	*Follow-up period* 3 months			1 year		
	'Stayers'	*'Leavers'*	*Total*	*'Stayers'*	*'Leavers'*	*Total*
AA	8	0	8	12	0	12
AB	6	0	6	1	0	1
BB	9	0	9	5	4	9
BC	2	0	2	1	1	2
CC	4	3	7	3	5	8
Totals	29	3	32	22	10	32

1. A = good outcome, B = average outcome, C = poor outcome.

psychodynamic groups before and felt that their approach to therapy was not affected. New members were added to replace drop-outs in all four groups but excluded from the research sample. One group had a planned closure at one year, while the other three continued into a second year.

Therapy

Group sessions were held on a weekly basis and lasted one-and-a-half hours. All eight therapists saw themselves as conducting an unstructured therapy in which patients were encouraged to explore their presenting problems and reflect upon relationships within the group. Further details of this approach are well described in Yalom's (1985) book. Bloch & Crouch (1985) describe this type of psychodynamic group therapy as 'interpersonal' and contrast it with an 'analytic' approach. The study did not include monitoring of the interventions therapists made within the therapy sessions, because checks of this kind were beyond the limited resources available.

Results

Table I shows the numbers of 'stayers' and 'leavers' at three months and one year and the therapists' ratings of their progress. Patients leave therapy for a variety of reasons, not always because they are unsatisfied. Three left within the first three months and all of these, according to their therapists, had not fitted in well and had not benefited. A further seven patients left therapy between three months and a year. Four of these were judged to have had an average outcome in that there had been some progress made and an agreement between the patient, the therapist and the group on the termination date.

It was found that the SCL 90 scores at three months and twelve months were both highly correlated with the initial SCL 90 scores ($r=0.89$ and $r=0.67$ respectively). Therefore it was necessary to take account of the dependence on initial scores in any analysis in which the SCL 90 was used as a measure of patient outcome. This was particularly important here because both the TEQ and C scores also showed significant correlations with the initial SCL 90 scores ($r=0.42$, $P<0.01$ in both cases). Adjustment for initial SCL 90 scores was effected via partial correlation and regression analyses. These showed a significant partial correlation between SCL 90 outcome at three months and TEQ ($P<0.01$) but no relationship with 'C' or DIQ. The regression analysis gave the following as the best predictor of SCL 90 (total symptom scores) at three months based on initial scores and the Claybury questionnaire:

$$\text{SCL 90 at three months} = -116 + 1.0 \times (\text{initial SCL 90}) + 2.8 \times \text{TEQ}$$

The correlation between the observed and predicted values was 0.92. At 12 months the regression equation was very similar but the contribution of the TEQ was no longer statistically significant, the *P-value rising from* $P=0.005$ at three months to $P=0.16$ at 12 months, even though its coefficient rose from 2.8 to 3.5. Failure to achieve significance was due to the greater variability of the 12 month values and the unavailability of SCL 90 scores from 10 patients, which reduced the effective sample size.

Table II shows the results of the correlations between the predictive and outcome measures. Symptom outcome on the SCL 90 was significantly predicted at three months only by the TEQ and therapists' initial rankings. There were no significant correlations with symptom outcome at 12 months, when only 22 patients could be included in the analysis. However, the correlations involving initial symptom score, TEQ and therapists' predictions were broadly similar to those at three months.

A similar picture emerged in the analysis of outcome based on therapists' assessments. These were of two types: the qualitative scores based on categories and the ranked indicator. Table II shows that both types of measure had significant correlations with therapists' predicted outcome as measured by their initial rankings. In addition, the 12 month outcomes showed significant positive correlations with TEQ ($r=0.54$ rankings, $r=0.47$ categories, $P<0.01$ in each case). At three months the correlations with TEQ, although present, were weaker and not significant. There were no significant correlations between outcome at three months or 12 months and either C or DIQ scores. The correlation between TEQ and the therapists' initial rankings was not large ($r=0.26$) and there was a significant partial correlation between 12 month outcome and TEQ after adjusting for the initial rankings. In other words, it

TABLE II
Correlations between predictive and outcome measures

Predictive measure	*Outcome measure*					
	SCL 90 symptom outcome[1]		*Therapists' categories*		*Therapists' rankings*	
	3 months	*12 months*	*3 months*	*12 months*	*3 months*	*12 months*
DIQ	−0.06	0.31	−0.14	0.02	0.00	0.03
TEQ	0.47*	0.34	0.23	0.47*	0.31	0.54*
'C'	0.28	−0.02	−0.06	0.20	0.13	0.25
Therapists' predictions (rankings)	0.40**	0.28	0.54*	0.51*	0.52*	0.50*
Therapists' predictions (categories)	0.26	0.03	0.44**	0.26 n.s.	0.41**	0.29

$^*P<0.01$, $^{**}P<0.05$.
1. Partial correlation coefficients, adjusted for initial SCL 90 scores.

seems that therapists' predictions can be improved upon if due attention is given to TEQ scores.

There was a reasonable measure of agreement between SCL 90 outcome and the two types of therapists' assessments as measured by appropriate partial correlation coefficients adjusted for initial SCL 90 scores ($r=0.52$ and 0.53 respectively at three months, $r=0.58$ and 0.35 at 12 months). All these coefficients were significant ($P<0.01$) except the last which related SCL 90 to the qualitative indicator at 12 months. At the same time it was clear that SCL 90 outcomes and therapists' assessments were not interchangeable.

Since the number of groups in the study was small (four) there was a danger that idiosyncratic events within one particular group may have had a large effect on the outcome. To assess this, the outcome results for each of the four groups were compared. There was little evidence of one group following a markedly different course from the other three. Each group contributed between two to three patients to the total of ten in the two lowest outcome categories (BC and CC) at one year, and either three or four patients to the two highest outcome categories (AA and AB, see Table I).

Of the three Claybury questionnaires, only the TEQ emerged as a significant predictor. The TEQ was subjected to an item analysis to see how each of the 15 items which contribute to the total score correlated with outcome. Therapists' ranked assessments at 12 months were chosen as the outcome measure since these showed the strongest correlation with the TEQ score. Table III shows that all of the 15 items correlated positively with this measure of outcome, with three individual items (19, 11, 9) showing

TABLE III
Correlations between TEQ items and therapists' ranked assessments at 12 months

TEQ item	*Content*	*Correlation*
19	I expect only my symptoms to change after treatment and not myself as a person.	0.59*
11	I can't see how other patients in treatment can help to cure anybody else except by chance or accident.	0.50*
9	Only a specialist in mental treatments will be able to help me get better.	0.40**
22	The doctor or therapist should always give direct advice to his patients.	0.33
6	Everbody's problems are different, so it wouldn't help me to discuss mine with other patients.	0.31
4	I don't understand how people can have difficulties in getting on with each other.	0.31
5	I don't think talking over emotional problems serves any useful purpose.	0.30
25	It is an important part of treatment for patients to believe that the doctors are all-powerful.	0.29
13	There would be much less mental illness if people exerted more control over themselves.	0.20
2	Just talking will never help me overcome my basic fears.	0.19
16	What I need is for the doctor or therapist to tell me what I am doing wrong and what I should do about it.	0.09
20	The personality of the therapist or doctor does not matter in the treatment, since it is his specialised knowledge that really counts.	0.09
27	A patient should not be expected to discuss really personal problems with the other patients.	0.09
10	I have never felt any pressing need to talk about my emotional problems.	0.05
21	Treatment is really being taught how to deal with difficult situations.	0.02

$^*P<0.01$, $^{**}P<0.05$.

significant correlations. The best prediction of outcome would have been obtained by adding items 19 and 11 together when a correlation of 0.70 is achieved, which is a better predictor than any combination of TEQ scores and therapists' predictions. Using the total TEQ score, the Claybury Selection Battery Manual (Caine *et al*, 1982) suggests that scores below 30 are predictive of positive outcome in small group psychotherapy based on the Caine & Wijesinghe (1976) study. The findings here are similar. A total of 13 patients began with TEQ scores below 30 and of these only one was given a poor therapist rating of outcome at 12 months.

The result of the second administration of the TEQ to the 22 stayers at one year was that there was little change in treatment expectancies regardless of outcome. For the 22 patients re-tested, the mean TEQ score at intake was 29.9 (s.d. 6.4) and the re-test score 29.2 (s.d. 6.9) with a correlation of 0.75. There was no correlation between the change in TEQ score and any outcome measure.

Discussion

In this study only one of the three Claybury questionnaires, the TEQ, showed significant correlations with outcome in small group psychodynamic therapy in the way predicted by the Claybury study. In fact, the TEQ is the only scale which addresses patients' attitudes to treatment directly.

Several patients wrote that they felt insulted by some of the questions on the 'C' questionnaire, which seem crude and outdated (e.g. questions about attitudes to strait-jackets, hippies, chaperones, etc.). Moreover, the high percentage of 'uncertain' responses suggested that many patients had problems with it.

Neither the 'C' scale nor the DIQ was found to correlate with patients' or therapists' ratings of outcome at either three months or one year, a result in conflict with the findings of Caine & Wijesinghe (1976). Differences in patients' personalities and initial symptoms, the type of therapy and the quality of therapist–patient relationships may all contribute to the discrepancies between the two studies. Nevertheless, the possibility remains that the 'C' and DIQ scales provide useful predictive information only in a small proportion of cases.

If these findings prove to be robust, group therapists may wish to consider using a simplified version of the Claybury battery, the TEQ alone, and further development of this questionnaire is indicated.

Acknowledgements

Thanks to Dr Marcia Davis and the Warwick MSc in Psychotherapy course 1984–1986, Joyce Rothschild, Brian Levy, Jim Kelman, Diana Palmer, and the staff and patients at the Uffculme Clinic.

References

BLOCH, S. & CROUCH, E. (1985) *Therapeutic Factors in Group Psychotherapy*. Oxford: Oxford University Press.

CAINE, T. M. & WIJESINGHE, O. B. A. (1976) Personality expectancies and group psychotherapy. *British Journal of Psychiatry*, **129**, 384–387.

——, —— & WINTER, D. A. (1981) *Personal Styles in Neurosis: Implications for Small Group Psychotherapy and Behaviour Therapy*. London: Routledge & Kegan Paul.

——, SMAIL, D. J., WIJESINGHE, O. B. A , *et al* (1982) *Manual for the Claybury Selection Battery*. Windsor, Berks: NFER Nelson.

DEROGATIS, L. R. (1981) *Description and Bibliography for the Symptom Checklist 90*. Clinical Psychometric Research Inc., PO Box 619, Riderwood, MD 21139 USA.

——, RICKELS, K. & ROCK, A. F. (1976) The SCL 90 and the MMPI: a step in the validication of a new self-report scale. *British Journal of Psychiatry*, **128**, 280–289.

GOLDSTEIN, A. P. (1962) *Therapist–Patient Expectancies in Psychotherapy*. New York: Pergamon Press.

KELLY, G. A. (1955) *The Psychology of Personal Constructs*. New York: Norton.

YALOM, I. D. (1985) *The Theory and Practice of Group Psychotherapy* (3rd edn). New York: Basic Books.

*Mark J. Pearson, MA, MRCPsych, MSc, *Consultant Psychotherapist, The Uffculme Clinic, Queensbridge Road, Moseley, Birmingham B13 8QD*, Alan J. Girling, MA, *Lecturer in Statistics, School of Mathematics and Statistics, University of Birmingham, Birmingham B15 2TT*

*Correspondence

British Journal of Psychiatry (1990), **157**, 389–391

Two-Year Follow-up of the Sheffield Psychotherapy Project

DAVID A. SHAPIRO and JENNY FIRTH-COZENS

Of 40 depressed and anxious patients who received treatment during the Sheffield Psychotherapy Project, 31 completed the BDI and SCL-90 at two-year follow-up. At both group and individual levels, outcomes were very similar to those obtained at the completion of treatment. Substantial minorities of patients reported major life events and seeking further help for psychological problems during the two years, but these reports were not reliably associated with symptoms at follow-up. Patients recalled very different elements in prescriptive and exploratory therapies as helpful, with a notable emphasis upon prescriptive therapy's relaxation and anxiety management as a source of problem solution.

Quantitative reviews of psychotherapy outcome show that treatments are generally effective (Smith *et al*, 1980; Shapiro & Shapiro, 1982). However, most studies have evaluated treatment effects immediately or at most a few months afterwards (Shapiro & Shapiro, 1983). In practical terms, such short-term effects are less impressive than longer lasting ones, the clinical significance being enhanced if patients maintain the gains shown during treatment.

However, Nicholson & Berman (1983) found that immediate and long-term effects of treatment were remarkably similar, whether compared at the level of the individual or the group, and therefore concluded that follow-up data are unlikely to be sufficiently informative to warrant the cost of their collection. On the other hand, Lambert *et al* (1986) suggested that follow-up data may be more informative in specific disorders, such as depression, that show a high relapse rate.

The practical significance of long-term follow-up data may be confounded by events in the client's life beyond the control or knowledge of the investigator. In particular, major life events and additional psychological therapy may exert effects that outweigh those of the treatment under investigation. It is therefore important to ask whether the patient's state at follow-up is related to either of these factors.

Clients' perceptions of the therapy are promising pointers to its effective ingredients (Orlinsky & Howard, 1986; Llewelyn *et al*, 1988). It is therefore of interest to compare client recollections at long-term follow-up of what was helpful in their treatment; if clients remember no differences between distinct treatments then it is unlikely that such treatments will differ in their long-term effectiveness.

We present two-year follow-up data from the Sheffield Psychotherapy Project (Shapiro & Firth, 1987), in which 40 patients with depression or anxiety received eight sessions of a behavioural/cognitive treatment, termed 'prescriptive therapy', and eight sessions of a psychodynamic treatment, termed 'exploratory therapy', in a crossover design with the same therapist throughout. Patients were assigned at random to receive either prescriptive or exploratory therapy in the first eight weeks. Outcomes were assessed using several interview and questionnaire measures, including the Beck Depression Inventory (BDI; Beck *et al*, 1961) and the Symptom Checklist–90 (SCL–90; Derogatis *et al*, 1973).

Changes during the two treatments were generally quite similar, although there was a slight advantage of prescriptive over exploratory therapy. Comparison between patients assigned to the two treatment groups in terms of changes from intake to the end of treatment and three-month follow-up showed no reliable differences, although therapists considered exploratory first to be preferable, and comments by clients supported this (Firth *et al*, 1986).

Method

The patients, therapists, treatments and assessment methods of the Sheffield Psychotherapy Project are described by Shapiro & Firth (1987). In the present study, we wrote to patients two years after completing treatment, asking their consent to send them a further questionnaire regarding their progress and their views on the treatment they had received. No patient who replied refused consent. Consenting patients received the BDI, the SCL–90, and a set of brief, open-ended questions which included what they thought the most helpful parts of the exploratory and prescriptive therapy were. Clients were also asked to list any major changes or events in their lives over the previous two years, and to state whether they had received help for psychological problems during that time.

We recognised the limitations of the instruments used (e.g. the life events method was primitive compared with that of Brown & Harris; 1978). However, we felt obliged to make only very limited demands upon patients, for three reasons: (a) we presented this additional request 'out of the blue' without seeking consent before treatment; (b) heavy demands had already been made on patients in completing process and outcome measures throughout therapy; and (c) a longer questionnaire would have deterred participation.

Results

Of the 40 patients completing treatment, 31 returned these questionnaires. Their mean age was 41.7 years, 17 were male and 15 had received prescriptive therapy first. At intake, 23 had received a CATEGO diagnosis of depression, and eight of anxiety. On none of the five intake measures reported by Shapiro & Firth (1987) did the mean score of those completing the two-year follow-up questionnaire significantly differ at intake from the mean of those not completing it (all t values < 1).

Table I shows means at intake, end of treatment, and two year follow-up on the BDI and SCL–90, together with the correlations of the two-year data with intake and end-of-treatment scores. The two-year means on both measures are almost identical to those obtained immediately after treatment. There were no differences on either measure at two-year follow-up between those diagnosed as depressed and those diagnosed as anxious at intake. The correlations between end-of-treatment and two-year data are substantial, and much higher than those between intake and two-year follow-up.

TABLE I

Means (s.d.) and r values on Beck Depression Inventory (BDI) and Symptom Checklist-90 (SCL–90) at intake, post-treatment and two-year follow-up

	Intake		*Post-treatment*		*Two-year follow-up*
Measure	*Mean (s.d.)*	r^1	*Mean (s.d.)*	r^1	*Mean (s.d.)*
BDI	20.9 (6.3)	0.20	8.4 (5.6)	0.55*	8.8 (7.9)
SCL–90	129.8 (42.8)	0.26	58.5 (40.5)	0.64*	58.4 (37.6)

*$P < 0.01$.

1. Correlation with follow-up data.

Of the 31 patients, 12 reported major life events (bereavement or severe illness within the family, divorce or separation, or job loss) and 13 reported receiving additional help (psychotherapy, counselling, or antidepressant medication). Patients were then divided into those who did and those who did not meet the criterion of a BDI score at follow-up of at least one standard deviation below the mean BDI score of the full sample of 40 patients at intake (Jacobson *et al*, 1984). On this criterion, 23 patients were designated 'improved' at follow-up, and the remaining eight patients were designated 'unimproved'. As shown in Table II, a slightly greater proportion of 'unimproved' than 'improved' patients reported experiencing major life events and seeking additional help for psychological problems. However, in neither case was this association statistically reliable.

TABLE II

Number of 'improved' and 'unimproved' patients who reported experiencing major life events and seeking further help for psychological problems at follow-up

	'Unimproved'[1] *patients*	*'Improved' patients*	*Total*
Event	4	8	12
No event	4	15	19
Total	8	23	31
Further help	5	8	13
No further help	3	15	18
Total	8	23	31

1. Unimproved = BDI score at follow-up of at least 1 s.d. below the mean BDI score of the 40 patients at intake.

Answers to the questions about what was most helpful about each therapy were classified following the scheme used by Llewelyn *et al* (1988). For prescriptive therapy, 30 of the 31 statements were classified as indicating 'problem solution', with 15 mentioning relaxation and anxiety management techniques as useful. In relation to exploratory therapy, only 19 scorable statements were made: seven indicated that the 'personal contact' was important; four showed that 'awareness' had been gained, and three 'insight'; two each suggested that the therapy had provided 'problem clarification' and 'understanding'; and one indicated 'problem solution'.

Discussion

The results suggest that the gains shown at the end of treatment were largely maintained over a two-year follow-up period, irrespective of whether the diagnosis was of depression or anxiety at intake. This supports the clinical importance of the changes reported by Shapiro & Firth (1987). However, 13 of the 31 clients did seek further treatment for various reasons, ranging from a quasi-recreational interest in further personal development, through specifically seeking marital therapy which was not provided by the Project, to treatment for an apparent relapse in symptoms.

The BDI and SCL–90 scores of individuals were quite stable over the two-year follow-up period, consistent with the findings of Nicholson & Berman (1983) and the proposition that follow-up scores add little information to that provided by post-treatment data, even for a predominantly depressed sample. The standard deviations indicate that the differences between these stable individual scores remained as large as they had been before, during, and immediately after treatment (Shapiro & Firth, 1987, Table I). The apparently modest effects of life events and further treatment on depression two years after therapy are consistent with a view of this as reflecting relatively stable personal characteristics.

However, within the limitations of the simple, open-ended questionnaire format used, the life event data suggest that for some individuals at least, life event stress may exacerbate or reactivate symptoms. The slight tendency for further treatment to be associated with poorer rather than better long-term outcome reflects these clients' need for help, rather than the further treatment bringing patients who seek it to a better psychological state than those who do not.

The sharp divergence between patients' recollections of helpful aspects of the two treatments closely resembles their accounts of these at the time (Llewelyn *et al*, 1988), and is consistent with their rating of prescriptive sessions as smoother and their mood as more positive after these sessions (Stiles *et al*, 1988). Taken together, these findings indicate that the quite similar immediate outcomes of the prescriptive and exploratory therapies, reported by Shapiro & Firth (1987), cannot be attributed to patients' failure to discern a difference between them. Longer-term differences in effectiveness between the therapies is examined in a one-year follow-up of the Second Sheffield Psychotherapy Project (Shapiro *et al*, 1990), in which each patient receives only one treatment.

The methodological limitations of this unanticipated and low-cost study must be acknowledged. For example, the impact of life events might have emerged more clearly using the interview method of Brown & Harris (1978). However, in view of the potential bias due to selective non-participation, this simple methodology was chosen in order to secure a reasonable participation rate, and the obtained figure of 77.5% was good for a two-year follow-up. In addition, we felt obliged to minimise the ethical costs of this further demand upon our patients. Finally, the present study demonstrates how low-cost follow-up methods can be applied in service settings with slender resources for research, such as the National Health Service.

References

BROWN, G. W., & HARRIS, T. (1978) *Social Origins of Depression*. London: Tavistock.

BECK, A. T., WARD, C. H., MENDELSON, M., *et al* (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–571

DEROGATIS, L. R., LIPMAN, R. S. & COVI, M. D. (1973) Symptom Checklist-90 (SCL-90), an out-patient rating scale: preliminary report *Psychopharmacology Bulletin*, **9**, 13–20.

FIRTH, J. A., SHAPIRO, D. A. & PARRY, G (1986) The impact of research on the practice of psychotherapy. *British Journal of Psychotherapy*, **2**, 169–179.

JACOBSON, N. S., FOLLETTE, W. C. & REVENSTORF, D. (1984) Psychotherapy outcome research: methods for reporting variability and evaluating clinical significance. *Behaviour Therapy*, **15**, 336–352.

LAMBERT, M. J., SHAPIRO, D. A. & BERGIN, A. E. (1986) The effectiveness of psychotherapy. In *Handbook of Psychotherapy and Behaviour Change* (3rd edn) (eds S. L. Garfield & A. E. Bergin), pp. 157–211. New York: Wiley.

LLEWELYN, S. P., ELLIOTT, R. K., SHAPIRO, D A., *et al* (1988) Client perceptions of significant events in prescriptive and exploratory periods of individual therapy. *British Journal of Clinical Psychology*, **27**, 105–114.

NICHOLSON, R. A., & BERMAN, J. A. (1983) Is follow-up necessary in evaluating psychotherapy? *Psychological Bulletin*, **93**, 261–278.

ORLINSKY, D. E. & HOWARD, K. I. (1986) The psychological interior of psychotherapy: explorations with the therapy session report. In *The Psychotherapeutic Process: A Research Handbook*. (eds L. S. Greenberg & W. M. Pinsof). New York: Guilford Press.

SHAPIRO, D. A. & SHAPIRO, D. (1982) Meta-analysis of comparative therapy outcome studies: a replication and refinement. *Psychological Bulletin*, **92**, 581–604.

——, & —— (1983) Comparative therapy outcome research. methodological implications of meta-analysis. *Journal of Consulting and Clinical Psychology*, **51**, 42–53.

—— & FIRTH, J (1987) Prescriptive v. exploratory psychotherapy: outcomes of the Sheffield Psychotherapy Project *British Journal of Psychiatry*, **151**, 790–799.

——, BARKHAM, M., HARDY, G. E. *et al* (1990) The second Sheffield Psychotherapy Project: rationale, design and preliminary outcome data. *British Journal of Medical Psychology*, **63**, 97–108.

SMITH, M. L., GLASS, G. V. & MILLER, T. I. (1980) *The Benefits of Psychotherapy*. Baltimore: Johns Hopkins University Press.

STILES, W. B., SHAPIRO, D. A. & FIRTH-COZENS, J. (1988) Do sessions of different treatments have different impacts? *Journal of Counseling Psychology*, **35**, 391–396.

*David A. Shapiro, BA, MSc, PhD, CPsychol, FBPsS, *Senior Scientist, MRC/ESRC Social and Applied Psychology Unit, University of Sheffield, Sheffield S10 2TN*; Jenny Firth-Cozens, MSc, PhD, CPsychol, *Research Fellow, Department of Psychology, University of Leeds*

*Correspondence

British Journal of Psychiatry (1990), 157, 392–398

A Systematic Inquiry into Recollections of Childhood Experience and their Relationship to Adult Defence Mechanisms

ELISABETH TAUSCHKE, HAROLD MERSKEY and EDWARD HELMES

The relationships between childhood experience, measured by the PBI, and adult defence mechanisms, evaluated through the DMI, were examined in 114 subjects. There were modest but significant correlations between the primitive defences of 'turning against others' and 'projection' on the one hand, and a lack of parental care, as experienced by the subjects, on the other. 'Principalisation' (e.g. intellectualisation, isolation and rationalisation) and 'reversal' (e.g. negation, reaction formation, denial and repression) were positively associated with parental care. These findings account for only a small portion of the variance but show that, as with certain personality traits, actual or perceived childhood experience contributes in a recognisable fashion to the patterns of adult defences and behaviour.

It is generally accepted that experiences in childhood are important in the development of personality. Adult defence mechanisms, for example, are presumed to be related to childhood experience.

Weinstock (1967) found that denial and repression in males were related to having a passive father in early childhood. Regression and doubt were related to the father's poor health and inactivity and his tendency to withdraw from conflict. Displacement seemed to be related to excessive parental restrictions, and projection to strain in the mother–child relationship at adolescence.

Vaillant (1974) found that "a subject's choice of defence mechanisms . . . seemed surprisingly independent of his childhood". He also remarks "men from warm childhoods might be able to attain good mental health without the benefit of mature defences, but men from bleak childhoods achieved good adult mental health only by deployment of mature defences" (Vaillant *et al*, 1986).

Both these studies are thorough and detailed. Weinstock's began in childhood, Vaillant's at the end of adolescence. Both authors take the view that certain defences are immature, but their selections are different. Weinstock regards denial, repression, regression and doubt as primitive mechanisms, unlike such mechanisms as projection that take place later. Vaillant considers projection, turning against self, hypochondriasis, fantasy (denial through fantasy) and acting out to be immature. Intellectualisation (isolation, undoing and rationalisation), repression, displacement (phobias, conversions), reaction formation, and dissociation (neurotic denial, counterphobias) were considered to be neurotic. Perry & Cooper (1989), using the Defense Mechanism Rating Scales, found support for Vaillant's model of a hierarchy of defences: those who had only neurotic defences had fewer symptoms and better global and psychosocial function than those with immature defences. Andrews *et al* (1989) have described the use of another questionnaire (Bond *et al*, 1983) to differentiate between mature defences, neurotic defences, and immature defences, these patterns varying with symptom scores.

However, the assessment of defence mechanisms itself is controversial (Bond *et al*, 1983; Vaillant, 1985; Vaillant *et al*, 1986). The main difficulties have been attributed to the fact that conceptually, defence mechanisms are an intrapsychic unconscious process that can only be appraised indirectly. Furthermore, there is much diversity in the definition, number and delineation of defence mechanisms, as well as in the importance ascribed to them (Haan, 1973; Suppes & Warren, 1975; Fraiberg, 1982; Beutel, 1985; Hoffmann & Martius, 1987). There are contradictory approaches in the hierarchical ranking of defences and insufficient separation of them from concepts of coping (Haan, 1963; Vaillant, 1971; Prystav, 1981; Battista, 1982; Vaillant *et al*, 1986). Vaillant allows some mature defences.

Tests developed to assess defence mechanisms include the Blacky Pictures (Blum, 1950), the Defense Mechanisms Test (Kragh, 1969) and scales by Haan (1965) based upon the Minnesota Multiphasic Personality Inventory. The Defense Mechanisms Inventory (DMI; Gleser & Ihilevich, 1969) has received the most attention in research.

The DMI is an objective paper-and-pencil test that measures the relative strength of five defence clusters. It comprises ten stories describing conflicts, relating authority, competition, independence, and sex. The subjects are asked to state their most likely actual behaviour, what they would most like to do, and their thoughts and feelings in response to each circumstance described. For each question the subjects have a choice of five answers in accordance with the five defensive scales, defined as: turning against the self (TAS), turning against the object (TAO, including identification with the aggressor and displacement), projection (PRO), reversal (REV, including negation, denial, reaction formation, and repression), and 'principalisation' (PRN, including intellectualisation, isolation and rationalisation).

The test–retest reliability of the DMI ranges between 0.48 and 0.93 for individual scales and between 0.59 and 0.84 for the test as a whole (Gleser & Ihilevich, 1969; Weissman *et al*, 1971; Woodrow, 1973; Rohsenow *et al*, 1978). Inter-item reliability is comparable (Juni, 1982; Vickers & Hervig, 1981). Content validity was assessed by comparing the DMI scores for behavioural alternatives with the judgements of clinicians (Gleser & Ihilevich, 1969). Raters agreed in 60% of their selections of one of the 15 possible defences to describe individual items of behaviour under TAS, REV and PRN. The results with PRN and TAO were less satisfactory. Blach & Fancher (1977) replicated these findings. Cramer (1988) notes that there is ample support for concurrent validity for the REV scale: in nine studies which compared this scale 11 times with scales from other tests, a significant relationship appeared. The status of the remaining three scales is moderately good. Many clinical and experimental studies have supported the validity and reliability of the DMI (Gleser & Ihilevich, 1969; Weissman *et al*, 1971; Ihilevich & Gleser, 1971; Gleser & Sacks, 1973; Blacha & Fancher, 1977; Juni & Masling, 1980; Cooper & Kline, 1982; Juni, 1982; Massong *et al*, 1982; Hoffmann & Martius, 1987).

There is some uncertainty with the REV scale, however, over whether it measures immature or mature defences. Cramer concludes that denial, repression, negation and reaction formation function by limiting awareness and are therefore immature defences. She sees this to be supported by the positive correlation of REV with field dependence, the tendency to overvalue one's performance, avoidance of sexual responses, and reduced dream recall. The positive correlation of REV with apparent psychological and physical health in several studies (Clum & Clum, 1973; Rohsenow *et al*, 1978; Wilson, 1982; Greenberg & Fisher, 1984; Peglar & Borgen, 1984) should be explained by the fact that the "use of this defence precludes recognizing or admitting the very existence of pathology, pain or discomfort on self-report measures" (Cramer, 1988). Her opinion is not in accordance with the theory of Vaillant (1976). According to his ranking, the defences summarised in REV are more mature than TAO, PRO or TAS.

According to Vaillant (1976) the only mature defences are sublimation, suppression, anticipation, altruism and humour. According to such a view, the DMI will not measure normal defences v. abnormal defences, but only greater or lesser degrees of immaturity. Conceptually, PRN and REV are held to measure defences that inhibit negatively toned emotionally expressive reactions. TAO and PRO allow negative expressions and turn them outwards, as opposed to TAS, which turns them inwards. In most studies, PRN and REV, and TAO and PRO are positively related to each other (Gleser & Ihilevich, 1969; Gleser & Sacks, 1973; Gur & Gur, 1975; Rohsenow *et al*, 1978; Vickers & Hervig, 1981; Passchier *et al*, 1984). Some authors (Juni & Masling, 1980; Juni, 1982; Wilson, 1982) therefore proposed that the DMI measures a continuum of defences relating to the expression of aggression, with defences that allow acting out at one end and those that inhibit hostile tendencies at the other. Cramer (1988) nevertheless found considerable evidence that the scales are assessing separate dimensions and should be individually maintained. She maintains that the positive correlation is additional evidence for the non-independence of the scales and that, also, the defence clusters might overlap in reality.

The DMI has an unusual feature in that the description of one type of behaviour, wish, thought or feeling reduces the score for other such categories that can be attributed to the subject. In other words it is an ipsative measure: the total score for any set of responses remains the same. Only the relative strength of defence mechanisms within individuals can be measured with accuracy (Angoff, 1971). This imposes some limitations on making correlative studies between DMI scores and between the DMI and other tests, but nevertheless they have been undertaken with a degree of success (Cooper & Kline, 1982).

So far as we are aware, defence mechanisms have not been related to previous childhood experiences in a systematic study, except by Weinstock and by Vaillant. If the DMI provides an independent measure of defences, then it would be worth relating it to another inventory, such as the Parental Bonding Instrument (PBI; Parker *et al*, 1979).

The PBI is a reliable and valid instrument for measuring the quality of parent–child relationships according to how the subject perceives it retrospectively. However, an independent relationship between the ratings of mothers' and subjects' reports has been obtained, as well as significant correlations between reported and observed parental characteristics (Parker & Lipscombe, 1981). Two major components are assessed: care v. indifference or rejection, and overprotection v. encouragement of independence. Associations have been shown between the scales of the PBI and depression (Parker, 1983*a*,*b*), hypochondriasis (Baker & Merskey, 1982), anxiety (Parker, 1981) and asthma (Parker & Lipscombe, 1979). The PBI has also shown some weak but significant correlations with the General Health Questionnaire and the Irritability/Depression and Anxiety Scale (Merskey *et al*, 1987).

The purpose of the present study was to examine whether any relationship could be demonstrated between childhood experience and characteristic defence mechanisms. If psychoanalytic hypotheses are correct, this should provide a systematic confirmation of them.

Method

Two consecutive groups of patients were asked to complete a set of forms. Only those patients were approached who were likely to be able to complete the tests. HM saw 115 patients: 60 of these were referred for a chronic pain problem and another 41 were attending the hospital open clinic. Demographic data collected included sex, age, marital status, and education.

Diagnoses for the referral group were made in accordance with DSM-III-R (American Psychiatric Association, 1987). For the clinic group, ICD-9 diagnoses were adapted to DSM-III-R by the crosswalk procedure (Thompson & Pincus, 1988).

Subjects completed the DMI and PBI and also the Hospital Anxiety and Depression Scale (HAD; Zigmond & Snaith, 1983) and the Hysteroid/Obsessoid Questionnaire (HOQ; Caine & Hope, 1967), not reported here. The PBI was administered in the modified version (Gamsa, 1987). Of 125 forms which were returned, 114 were usable.

Statistics

Comparisons between groups were performed using multivariate analyses of variance (MANOVA), supplemented by univariate analyses. Correlations were calculated using Pearson's coefficient. All calculations were performed using SYSTAT (Wilkinson, 1988). The ipsative nature of the DMI was adjusted using two methods. In the first, raw DMI scores were converted to standard scores using the norms of Gleser & Ihilevich (1969). This conversion results in a 'normative ipsative' score (Hicks, 1970) that is not formally ipsative, in that the sum of scores for each person is not a constant. In the second, adapted from Cooper & Kline (1982), the upper and lower 25% of the score distribution were found for each DMI scale and the middle 50% discarded. Scores from the high- and low-scoring patients were then correlated or cross-tabulated with the other measures.

Results

One hundred and fourteen sets of tests were evaluated. Eleven patients were excluded from the study, two because they had difficulties with the English language, and nine because they failed to complete enough of the measures. Two of the patients excluded had been referred for chronic pain.

The 114 patients comprised 74 women and 40 men (average age 44.8 years, range 21–78). Sixty-seven patients were married, 26 single, 11 divorced, 4 separated, 6 widowed. Diagnostic groupings were: mood/anxiety disorder, 57 patients; personality disorder, 11; schizophrenia, 6; no current psychiatric disorder, 40 (27 with chronic pain, 13 with psychiatric disorders in remission).

TABLE I

Mean scores of 114 subjects on the Parental Bonding Instrument and Defense Mechanisms Inventory

	Mean	*s.d.*
PBI		
Maternal care	21.62	11.22
Paternal care	18.09	10.74
Maternal overprotection	13.84	7.90
Paternal overprotection	13.14	7.97
DMI		
TAO	33.39	9.25
TAS	41.15	7.49
PRO	36.80	5.88
PRN	45.45	7.28
REV	43.18	8.32

TABLE II

Correlations within the Parental Bonding Instrument and Defense Mechanisms Inventory

	r	P
PBI		
Maternal care/maternal overprotection	−0.458	0.010
Maternal care/paternal care	0.425	0.000
Maternal care/paternal overprotection	−0.202	0.040
Paternal care/paternal overprotection	−0.314	0.001
Paternal care/maternal overprotection	−0.102	0.304
Paternal overprotection/maternal overprotection	0.519	0.000
DMI		
TAO/TAS	−0.400	0.000
TAO/PRO	0.496	0.000
TAO/PRN	−0.578	0.000
TAO/REV	−0.625	0.000
TAS/PRO	−0.295	0.002
TAS/PRN	−0.222	0.024
TAS/REV	−0.066	0.508
PRO/PRN	−0.427	0.000
PRO/REV	−0.636	0.000
PRN/REV	0.285	0.003

The mean scores and standard deviations for all the tests are shown in Table I. Scores on the PBI for overprotection are high, and scores for care tend to be lower than those from normal populations. There are no noteworthy differences from normal populations on the DMI scales.

Table II shows the correlations within the tests. These are all in the expected direction. For the PBI, both maternal and paternal care are negatively correlated with overprotection, which is how the test was designed. For the DMI, the inter-relations of the scales are in the same directions as described in the introduction. We found consistently positive inter-relations between scores on TAO and PRO and on REV and PRN.

No correlation was found between age and any DMI scale. TAO was positively ($P=0.006$) and REV was negatively ($P=0.016$) related to education. (Rohsenow *et al* (1978) found TAS and REV negatively correlated to education, PRN positively and TAO and PRO unrelated to education; they found that greater intelligence was related positively with TAO and negatively with REV and TAS.)

Table III shows the scores for women and men. The MANOVA on the PBI scales was not significant for sex differences (Pillai's trace=0.065, d.f. 4, 103, $P>0.10$). The corresponding MANOVA for the DMI scales was significant for sex differences (Pillai's trace=0.159, d.f. 4, 103, $P<0.005$). The univariate tests showed that females scored lower on maternal care ($P=0.037$) and REV ($P=0.034$) and higher on TAS ($P=0.001$). (The significantly higher tendency for women to blame themselves has been shown in a number of studies (Gleser & Ihilevich, 1969; Bogo *et al*, 1970; Weissmann *et al*, 1971; Gleser & Sacks, 1973; Massong *et al*, 1982).

The score on maternal care for our women is not only significantly lower than for our male sample but is also lower than the general practice sample of Parker *et al* (1979). This might be an indication of the selected population of our study, and indeed, there are significant differences between the women here and those in Parker's (1983*a*) depressed sample (Table IV). The male population in comparison shows no differences for parental care compared with Parker's general practice sample. With the DMI, no such differences could be found, neither for our women nor for the men when compared with the psychiatric out-patient sample of Gleser & Ihilevich (1969).

No correlations with overprotection for any measure of the DMI were significant at the level $P=0.05$. PRN correlated negatively ($P=0.082$) with paternal overprotection. However, significant correlations between the PBI and the DMI appeared with the care scales for both parents. Using the normative ipsative DMI scales, maternal care is negatively associated with TAO ($r=-0.288$, $P=0.003$) and positively related with PRN ($r=0.216$, $P=0.027$) and REV ($r=0.212$, $P=0.031$). Paternal care shows negative correlations with TAO ($r=-0.194$, $P=0.049$) and PRO ($r=-0.243$, $P=0.013$) and positive correlations with REV ($r=0.236$, $P=0.007$). Five of the ten correlations between parental care and the DMI scales are significant at $P<0.05$ using the fully ipsative measures. Analyses with even more conservative methods led to the same pattern of correlation.

TABLE III

Sex differences: mean values with the Parental Bonding Instrument and the Defense Mechanisms Inventory

	Women (*n*=*74*)	*Men* (*n*=*40*)	*P*
Maternal care	20.25	24.1	0.037
Paternal care	18.41	20.02	NS
Maternal overprotection	14.61	12.47	NS
Paternal overprotection	14.14	11.35	NS
TAS	42.45	38.80	0.001
REV	42.38	44.60	0.034
TAO	32.56	34.92	NS
PRO	36.80	36.82	NS
PRN	45.79	44.82	NS

Discussion

Our study was done on a selected population, but since we were examining correlations between scales the selection bias may not be relevant. The patients were perhaps more heterogeneous than a psychiatric sample from one source, or with one diagnosis, but more homogeneous than overt patient and normal control groups. Having almost twice as many women as men is common in a psychiatric population.

The finding of a negative correlation of REV and a positive one of TAO with education is similar to

TABLE IV

Comparison of the present female sample with depressed and general practice female samples (Parker, 1983a)

	A: present sample	*B: Parker depressed*	*C: Parker GP*	$t_{A/B}$	$t_{A/C}$
Maternal care	20.5	19.5	27.1	0.00	−5.00**
Paternal care	18.4	19.1	24.3	−1.28	−5.03**
Maternal overprotection	14.6	18.2	12.9	−2.37	1.79*
Paternal overprotection	14.1	15.5	12.7	0.07	1.47*

*$P<0.05$, **$P<0.01$.

that of Rohsenow *et al* (1978) but opposite to *a priori* expectation. In our sample the more 'normal' individuals psychologically had chronic pain. Many were manual workers and they had 11.8 years of education. Here the association of TAO and REV with education may be an artifact of selection. The matter remains open. The correlations found within the tests suggest that for the most part they are functioning in accordance with previous reports in the literature.

The main purpose of this paper was to explore the relationship between the PBI and DMI. Here it is striking but not surprising that overprotection showed little relationship with the defence mechanisms examined. In general, overprotection has been found to be strongly associated with hypochondriasis (Baker & Merskey, 1982) and somewhat with anxiety (Parker, 1981; Merskey *et al*, 1987) and with irritability (Merskey *et al*, 1987). Correlations between overprotection and painful conditions as such appeared to be absent (Salter *et al*, 1983; Merskey *et al*, 1985). But the overprotection scales seem to be not as strong as the care scale, and indeed Parker (1983*b*) stated that the overprotection scales are less homogeneous than the care scales, and the defence of hypochondriasis is not measured in the DMI.

The relationships between both maternal and paternal care and the DMI are similar and consistent, with only minor differences. TAO shows a negative relationship with both paternal and maternal care. PRO showed modest negative association with maternal care and a significant negative association with paternal care. This can be interpreted to mean that adults who believe they lacked care as children tend to blame others and are also more aggressive towards others.

TAS shows no relationship with any of the measurements of the PBI. Nevertheless, it has a definite pattern associated with personality features both in some of our additional data and in other studies (Bogo *et al*, 1970; Donovan *et al*, 1977; Greenberg & Fisher, 1984; Ihilevich & Gleser, 1971; Rohsenow *et al*, 1978; Scholz, 1973; Schueler *et al*, 1982). This measure can be effective but has no close relationship with phenomena assessed by the PBI.

PRN and REV tend to be associated with each other in this as in other studies. Curiously, at first sight, good care seems to lead to an increase in the mechanisms of denial and repression. We might ask if this makes sense. If the DMI is treated as an ipsative test, some measures must fall as others rise. Thus a negative relationship of REV and PRN to TAO and PRO would seem to leave TAS in a neutral position, while pushing REV and PRN in the opposite direction to TAO and PRO. The increased positive relationship of REV and PRN with care might then be an artifact. However, reducing the ipsative features as we have done does not change the relationship. Hence PRN and REV probably have a real relationship with care.

If the DMI had incorporated the sort of defences which Vaillant considers to be mature defences (sublimation, suppression, humour, etc.) it is possible that better care would have demonstrated a preferential association with supposedly superior methods of defence. Lacking such a dimension, the DMI nevertheless shows that REV and PRN, which are thought to be more mature defences, are positively associated with care, as opposed to TAO and PRO, which are negatively related to care. Taking the positive relationships between measures of psychological and physical health in Vaillant's studies at face value would argue against Cramer's conclusion that REV summarises primitive defences. Both Vaillant's ranking and our own results confirm this. Maternal care is more strongly associated with the reduction of primitive and aggressive behaviour towards others (TAO) than is paternal care, although both measures of care operate in the same direction. The mean values for maternal care are also slightly higher for mothers than for fathers. This suggests that maternal care is more important in reducing defences of aggression and hostility. Children who enjoy their mothers' love act kindly to others.

Paternal attention tends to be accompanied by demands for improved behaviour. In meeting the paternal standards, PRN and REV will be employed as modes with which to manage paternal pressures. In a caring family with steady discipline, children learn not to blame or attack others unduly and will be disposed to protect themselves by the relatively mature defences of REV and PRN in the face of discipline.

Acknowledgements

Elisabeth Tauschke was supported by a research fellowship from the FAZIT-Stiftung, Federal Republic of Germany. We thank Dr Viera Barta and the staff of the Mental Health Clinic at London Psychiatric Hospital, who selected and diagnosed the patients in the clinic series, and Ms Mai Why, MLS, who provided extensive bibliographical help.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA

Andrews, G., Pollock, C. & Stewart, G. (1989) The determination of defense style by questionnaire. *Archives of General Psychiatry*, **46**, 455–460.

Angoff, W. H. (1971) Scales, norms and equivalent scores. In *Educational Measurement* (2nd edn) (ed. R. L. Thorndike). Washington, DC: American Council on Education.

Baker, B. & Merskey, H. (1982) Parental representations of hypochondriacal patients from a psychiatric hospital. *British Journal of Psychiatry*, **141**, 233–238.

Battista, J. R. (1982) Empirical test of Vaillant's hierarchy of ego functions. *American Journal of Psychiatry*, **139**, 356–357.

Beutel, M. (1985) Zur Erforschung der Verarbeitung chronischer Krankheit. *Psychotherapie, Psychosomatik und Medizinische Psychologie*, **35**, 295–301.

Blacha, M. D. & Fancher, R. E. (1977) A content validity study of the Defense Mechanisms Inventory. *Journal of Personality Assessment*, **41**, 402–404.

Blum, G. S. S. (1950) *The Blacky Pictures*. New York: Psychological Corporation.

Bogo, N., Winget, C. & Gleser, G. C. (1970) Ego defences and perceptual style. *Perceptual and Motor Skills*, **30**, 599–604.

Bond, M., Gardner, S., Christian, J., *et al* (1983) Empirical study of self-rated defence styles. *Archives of General Psychiatry*, **40**, 333–338.

Caine, T. M. & Hope, K. (1967) *Manual of the Hysteroid/Obsessoid Questionnaire*. London: University of London Press.

Clum, G. A. & Clum, J. (1973) Choice of defense mechanisms and their relationship to mood level. *Psychological Reports*, **32**, 507–510.

Cooper, W. H. & Kline, P. (1982) A validation of the Defence Mechanism Inventory. *British Journal of Medical Psychology*, **55**, 209–214.

Cramer, P. (1988) The Defense Mechanisms Inventory: a review of research and discussion of the scales. *Journal of Personality Assessment*, **52**, 142–164.

Donovan, D. M., Rohsenow, D. J., Schau, E. J., *et al* (1977) Defensive style in alcoholics and nonalcoholics. *Journal of Studies on Alcoholism*, **38**, 465–470.

Fraiberg, S. (1982) Pathologic defenses in infancy. *Psychoanalytic Quarterly*, **51**, 612–635.

Gamsa, A. (1987) A note on a modification of the Parental Bonding Instrument. *British Journal of Medical Psychology*, **60**, 291–294.

Gleser, G. C. & Ihilevich, D. (1969) An objective instrument for measuring defense mechanisms. *Journal of Consulting and Clinical Psychology*, **33**, 51–60.

—— & Sacks, M. (1973) Ego defenses and reaction to stress: a validation study of the Defense Mechanisms Inventory. *Journal of Consulting and Clinical Psychology*, **40**, 181–187.

Greenberg, S. P. & Fisher, S. (1984) Menstrual discomfort, psychological defense, and feminine identification. *Journal of Personality Assessment*, **48**, 643–648.

Gur, R. E. & Gur, R. C. (1975) Defense mechanisms, psychosomatic symptomatology and conjugate lateral eye movements. *Journal of Consulting and Clinical Psychology*, **43**, 416–420.

Haan, N. (1963) Proposed model of ego functioning: coping and defence mechanisms in relationship to IQ change. *Psychological Monographs*, **77**, 1–23.

—— (1965) Coping and defense mechanisms related to personality inventories. *Journal of Consulting Psychology*, **29**, 373–378.

—— (1973) Defence, displacement and the ego's algebra. *International Journal of Psychoanalysis*, **54**, 247–257.

Hicks, L. E. (1970) Some properties of ipsative, normative, and forced-choice normative measures. *Psychological Bulletin*, **74**, 167–184.

Hoffmann, S. O. & Martius, B. (1987) Zur testdiagnostischen Erfassung der Abwehrstrukturen von Patienten mit Angstneurosen, paranoiden Syndromen und karzinomatoesen Erkrankungen. *Psychotherapie, Psychosomatik und Medizinische Psychologie*, **37**, 97–104.

Ihilevich, D. & Gleser, G. C. (1971) Relationship of defense mechanisms to field dependence–independence. *Journal of Abnormal Psychology*, **77**, 296–302.

Juni, S. (1982) The composite measure of the Defense Mechanisms Inventory. *Journal of Research in Personality*, **16**, 193–200.

—— & Masling, J. (1980) Reaction to aggression and the Defense Mechanisms Inventory. *Journal of Personality Assessment*, **44**, 484–486.

—— & Yanishevsky, D. S. (1983) Defensive style: state or trait? *Journal of Personality Assessment*, **47**, 536–538.

Kragh, U. (1969) *The Defense Mechanism Test (DMT)*. Stockholm, Sweden: Testfoerlaget.

Massong, S. R., Dickson, A. I., Ritzler, B. A., *et al* (1982) A comparison of defense mechanism measures: the Defense Mechanisms Inventory and the Blacky Defense Preference Inventory. *Journal of Personality Assessment*, **46**, 477–480.

Merskey, H., Brown, A., Malhotra, L., *et al* (1985) Psychological normality and abnormality in persistent headache patients. *Pain*, **23**, 35–47.

——, Lau, C. L., Russell, E. S., *et al* (1987) Screening for psychiatric morbidity. The pattern of psychological illness and premorbid characteristics in four chronic pain populations. *Pain*, **30**, 141–157.

Parker, G. (1981) Parental representations of patients with anxiety neurosis. *Acta Psychiatrica Scandinavica*, **63**, 33–36.

—— (1983*a*) Parental 'affectionless control' as an antecedent to adult depression. *Archives of General Psychiatry*, **40**, 956–960.

—— (1983*b*) *Parental Overprotection*. New York: Grune & Stratton.

—— & Lipscombe, P. (1979) Parental overprotection and asthma. *Journal of Psychosomatic Research*, **23**, 295–299.

—— & —— (1981) Influences on maternal overprotection. *British Journal of Psychiatry*, **138**, 303–311.

——, Tupling, H. & Brown, L. B. (1979) A Parental Bonding Instrument. *British Journal of Medical Psychology*, **52**, 1–10.

Passchier, J., van der Helm-Hylkema, H. & Orlebeke, J. F. (1984) Personality and headache type: a controlled study. *Headache*, **24**, 140–146.

Peglar, M. & Borgen, F. H. (1985) The defense mechanisms of coronary patients. *Journal of Clinical Psychology*, **40**, 669–679.

Perry, J. C. & Cooper, S. H. (1989) An empirical study of defense mechanisms. I. Clinical interview and life vignette ratings. *Archives of General Psychiatry*, **46**, 444–452.

Prystav, G. (1981) Psychologische Copingforschung: Konzeptbildungen, Operationalisierungen und Messinstrumente. *Diagnostica*, **27**, 189–214.

Rohsenow, D. J., Erickson, R. C. & O'Leary, M. R. (1978) The Defense Mechanisms Inventory and alcoholics. *International Journal of Addiction*, **13**, 403–414.

Salter, M., Brooke, R. I., Merskey, H., *et al* (1983) Is the temporo-mandibular pain and dysfunction syndrome a disorder of the mind? *Pain*, **17**, 151–166.

Scholz, J. A. (1973) Defence style in suicide attempters. *Journal of Consulting and Clinical Psychology*, **40**, 70–73.

Schuebler, D. E., Herron, W. G., Poland, H. V., *et al* (1982) Defence mechanisms in reactive and process schizophrenia. *Journal of Clinical Psychology*, **38**, 486–489.

Suppes, P. & Warren, H. (1975) On the generation and classification of defence mechanisms. *International Journal of Psychoanalysis*, **56**, 405–414.

Thompson, J. W. & Pincus, H. A. (1988) *Crosswalk*. Washington, DC: American Medical Records Association, Office of Research, APA.

Vaillant, G. (1971) Theoretical hierarchy of adaptive ego mechanisms. *Archives of General Psychiatry*, **24**, 107–118.

—— (1974) Natural history of male psychological health. II. Some antecedents of healthy adult adjustment. *Archives of General Psychiatry*, **31**, 15–22.

—— (1976) Natural history of male psychological health. V: The relation of choice of ego mechanisms of defence to adult adjustment. *Archives of General Psychiatry*, **33**, 535–545.

—— (1985) Maturity of ego defences in relation to DSM-III axis II personality disorder. *Archives of General Psychiatry*, **42**, 597–601.

——, Bond, M. & Vaillant, C. (1986) An empirically validated hierarchy of defence mechanisms. *Archives of General Psychiatry*, **43**, 768–794.

Vickers, R. R. & Hervig, L. K. (1981) Comparison of three psychological defense mechanism questionnaires. *Journal of Personality Assessment*, **45**, 630–638.

Weinstock, A. R. (1967) Family environment and the development of defense and coping mechanisms. *Journal of Personality and Social Psychology*, **5**, 67–75.

Weissman, H. N., Ritter, K. & Gordon, R. M. (1971) Reliability study of the Defense Mechanisms Inventory. *Psychological Reports*, **29**, 1237–1238.

Wilkinson, L. (1988) SYSTAT: the system for statistics. Evanston, Illinois: SYSTAT, Inc.

Wilson, J. F. (1982) Recovery from surgery and scores on the Defense Mechanisms Inventory. *Journal of Personality Assessment*, **46**, 312–319.

Woodrow, J. Z. (1973) A factor analysis and revision of the Defense Mechanisms Inventory. PhD thesis, Ohio University (University microfilm no. 7325759). Cit. *Defense Mechanisms* (1986; P. Ihilevich & G. C. Gleser). Owosso, Michigan: DMI Associates.

Zigmond, A. S. & Snaith, R. P. (1983) The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, **67**, 361–370.

Elisabeth Tauschke, MD, *Research Fellow London Psychiatric Hospital*; *Harold Merskey, DM, FRCP, FRCP(C), FRFCPsych, FAPA, *Director of Research, London Psychiatric Hospital, Professor of Psychiatry, University of Western Ontario*; Edward Helmes, PhD, *Clinical Associate Professor, Department of Psychiatry, University of Western Ontario*

*Correspondence: *Department of Research, London Psychiatric Hospital, 850 Highbury Avenue, PO Box 2532, Terminal 'A', London, Ontario N6A 4H1, Canada*

British Journal of Psychiatry (1990), 157, 399–403

Incest, Incestuous Fantasy & Indecency
A Clinical Catchment Area Study of Normal-Weight Bulimic Women

J. HUBERT LACEY

This systematic study of 112 consecutive referrals of normal-weight bulimic women from a circumscribed urban catchment area found that eight patients (7%) reported sexual abuse involving physical contact. Four (3.6%) of these described incest, but only in two cases (1.8%) did the incest occur in childhood. Just over 5% of the sample reported unlawful sexual intercourse in childhood or gross indecency. Eighteen additional patients reported incestuous fantasies and two of these were partly acted out. The study indicates that incest and sexual abuse reported by bulimics is usually in those multi-impulsive patients who also abuse alcohol or drugs as well as food. The reported prevalences of incest and indecency in normal-weight bulimic women are only slightly higher than figures derived from general population surveys.

Recently, both childhood sexual abuse (CSA) and incest, whether involving children or adults, have received substantial publicity. The widespread view gained is that their incidences are common and increasing in frequency. However, data are scarce. The information which is available is open to criticism particularly because of methodology and case definition. The problem is often compounded by misquotation or confusion about terms (Markowe, 1988).

Although the methods of the MORI survey were excellent (Baker & Duncan, 1985), the definition of sexual abuse used in it was broadly drawn and this is perhaps why the study reported a higher figure of female childhood sexual abuse (12%) than that in the British Broadcasting Corporation's (BBC; quoted by Markowe, 1988) Childwatch interview survey (4%). More specifically, MORI reports vaginal sexual intercourse by 0.6% of the sample and the BBC by 0.4%, although in the latter survey, a further 2.2% of women reported that intercourse was attempted in childhood and a similar percentage (although not necessarily additive) were "forced to do something sexual". What is, however, quite clear from these and other studies (Finkelhor, 1979; Mrazek *et al*, 1983; Wyatt, 1985; Nash & West, 1985; Mullen *et al*, 1988) is that much sexual abuse never reaches the attention of professionals; that most studies underestimate, however the cases are defined; that much abuse is non-physical and often experienced on a single occasion; and that intercourse and attempted intercourse represents only a minority of reported childhood sexual experiences.

Although regarded as an offence against God, incest did not become a crime in England until 1908. Bluglass (1979) outlined the legal and clinical aspects. It is an offence for a man to have sexual intercourse with a woman whom he knows to be his granddaughter, daughter, sister (or half-sister) or mother. Both sexual offences against children and incest are separate offences in law, and 'additive', that is, there are defined offences against children which stand irrespective of any relationship between adult and child and, additionally, where there is a 'blood' relationship between the two which is legally defined such as to make the contact incestuous, then the latter offence is added to the former. Sexual offences against children include "unlawful sexual intercourse" with a girl under 16 years, or with a girl under 13 years (different rules of liability apply in these two cases), and indecency with children. This latter offence involves inviting or inciting a child to perform acts of gross indecency upon an adult.

Legal definitions do not, of course, consider the psychological and interactive aspects of child sexual abuse. One of the most widely referenced definitions given by Schechter & Roberge (1976) refers to sexual exploitation of children as "the involvement of dependent, developmentally immature children and adolescents in sexual activities that they do not fully comprehend, are unable to give informed consent to, and that violate the social taboos of family roles".

Descriptions of incest and CSA by the eating disordered are not new (Abraham & Beaumont, 1982; Crisp, 1984), but recently the opinion has gained ground at a clinical level that these events are commonly reported by such patients. Oppenheimer *et al* (1985) systematically studied 78 eating-disordered patients of mixed diagnoses. Nearly two-thirds of the women patients described adverse sexual experiences, mostly in childhood, and 36% of the perpetrators were members of the patients' families.

Sloan & Leichner (1986) report that 39% of the anorexic and bulimic patients admitted to their unit had a history of sexual abuse. The association has gone further and the view has been expressed that there is a relationship between sexual abuse and subsequent disturbed eating (Calan & Slade, 1987). Such a claim and the disturbingly high figures indicated above warrant further examination in a representative catchment area population. Eating disorders occur in a heterogenous population and specific inquiry among normal-weight bulimic women is needed.

Method

All the patients in this series were interviewed by the author and were under his consultant care. They were a consecutive series of 112 female patients who attended the clinic during a 40-month period. All the patients entering the study were diagnosed by the author as having bulimia according to the criteria of DSM-III-R (American Psychiatric Association, 1987), but any patient who had anorexia nervosa (Crisp, 1967) or massive obesity (over 50% of mean-matched population weight) was excluded. All patients lived in the Wandsworth & Merton District Health Authority area and all had been referred by their general practitioner.

The study was done as a part of a larger clinical descriptive study (Lacey, 1987). All patients were asked to attend for a three-hour assessment interview and were accompanied by an informant who was usually the patient's mother. A standardised psychiatric interview was done although extra emphasis was placed on those clinical and biographical features which are often found in the eating disorders. The author was guided by a semi-structured history sheet, devised by himself, so that the interviews could be done in the same order and had consistency (Lacey, 1987).

Towards the end of the clinical interview, a detailed sexual history was taken. The questions were designed not only to record experiences but also to determine the pattern of sexual development. Thus questions were asked, in order, on bodily development and associated embarrassment; onset of sexual feelings; the first boyfriend and details of subsequent emotional relationships; clinical and demographic data on the current sexual partner, pregnancies and children. The patient was then asked about the extent of her sexual experiences including vaginal, oral and anal intercourse, homosexuality and rape. Only then were incestuous feelings and incest broached. The patient was told not to answer the question immediately but to think carefully for thirty seconds and only then reply.

Results

Eight patients reported incest or sexual abuse: five were with their natural fathers (cases 1, 49, 55, 88 and 96) and the others with respectively a grandfather (case 31), mother (case 75) and stepfather (case 94). Five patients reported vaginal intercourse (cases 1, 33, 88, 94 and 96), two reported fellatio (cases 1 and 88) and five were instructed to masturbate their partner (cases 1, 49, 55, 75 and 88). One (case 94) became pregnant and had a therapeutic abortion. Details are given in the Appendix.

Of the five perpetrators who were the patients' natural fathers, four (cases 1, 55, 88 and 96) and case 94's stepfather had histories of alcohol abuse – as subsequently did their victims. Most perpetrators continued to have concurrent relationships with their wives and involve other daughters. There was also a history of violence, irritability and criminal offences. Cases 31, 49 and 75 were quite different, however. Here, the patients described gradual seductions at a time when the perpetrators' usual sexual partners were absent by separation or illness. The two men were primarily inadequate rather than aggressive but the partner of case 75, who was her mother, appeared to have been ill. The mother was interviewed: there was no current psychosis but it was clear that there had been a profound depression with 'biological' features.

Demographic and general clinical data are given in Table I. Sexually abused bulimics are older than others in the clinic (28 years; 24.8 years), less likely to induce vomiting, but more likely to abuse laxatives and engage in other self-damaging behaviours. Thus, six of these eight patients described alcohol (cases 55, 88, 94 and 96) or drug abuse (cases 31 and 75) in addition to their eating disorders. One was diagnosed alcoholic (case 94) and one used heroin (case 75). Six reported repeated stealing.

Five of the eight patients reported poor or indifferent relationships with their school teachers; three (cases 55, 88 and 96) reported that they repeatedly questioned the teachers' authority and were disciplined by them. Two reported that they were generally apathetic about school and the third felt that she was 'picked on' by her teachers and demonstrated lack of respect. Six of the eight cases reported repeated truancy. Five reported either poor or indifferent relationships with their peers and particularly mentioned that they had few friends or superficial friendships, or found it difficult to mix and described underdeveloped social skills.

The sexual histories of these eight patients showed some differences from other patients in the Bulimic clinic, although numbers are too small for statistical comparisons. While the average age of onset of sexual feelings was 13.5 years in the general bulimia clinic population, all these patients described libido before the age of 12 years. The victims reported first sexual intercourse, other than with the perpetrator, at an average age of 15 years while the average age for the rest of the clinic population was 17.6 years. While 18 of the 112 in the clinic population reported having 20 or more sexual partners, this included all eight cases in this study. No victim had engaged in prostitution, although four non-abused women had. Only one of the abused cases reported a homosexual relationship, describing it as "calming, kind and comfortable". It took place over a two-month period during a time of sexual disinhibition. Two victims reported rape (see Appendix).

Eighteen additional patients reported incestuous feelings or fantasies which were felt by the patient to be important. In 15 cases the fantasy involved their fathers, in two cases their mothers and in one case a brother. For two, the fantasies were in part acted out. Thus case 46 reported that after her parents divorced, her father remarried a woman

TABLE I
Demographic and clinical features

Features	*Case numbers* *1*	*31*	*49*	*55*	*75*	*88*	*94*	*96*
Age of presentation: years	28¾	21¾	30	30½	21¾	30¼	26	34½
Marital status[1]	S	S	D	S	S	M	S	S
% MMPW[2]	109%	88%	105%	113%	105%	110%	91%	96%
Age of onset of binge-eating: years	20¾	17½	19	22	15½	12¾	13½	21
Age of onset of vomiting: years	23½	17	–	–	–	–	20	21½
Laxative abuse	Bi-weekly	Daily	Daily	Daily	Daily	Daily	Daily	Daily
Alcohol – use and abuse[3]	Social	Social	Social	Abuse	Abstain	Abuse	Abuse	Abuse
'Street drug' use	No	Yes	No	No	Yes	No	No	No
History of repeated stealing	Yes	No	Yes	Yes	Yes	Yes	Yes	No
History of overdosing	No	No	No	No	No	No	>2×	>2×

1. S = single, M = married, D = divorced.
2. % mean-matched population weight.
3. Abstain = 0 units/week; social = 1–20 units/week; abuse = 36+ units/week.

TABLE II
Cases reporting sexual abuse

Nature of sexual offence	*Case numbers*	*Total*
Unlawful sexual intercourse with patient when under 13 years	88	1
Unlawful sexual intercourse with patient when under 16 years	31, 94	2
Indecency with physical contact	1, 31, 49, 55, 88, 94	6
Incest	31, 75[1], 88, 96[1]	4

1. As an adult only.

of the same christian name as herself. The new stepmother died shortly afterwards and her wedding-ring was removed and given by her father to the patient who then wore it on her wedding finger. Case 109 was one of two adopted daughters. Her father had many affairs and eventually left his wife for one of them. A quite bizarre situation then developed whereby all four women (that is, wife, the two adopted daughters and the mistress) lived with him and "competed for my father's attention". He organised a slimming competition for all four, the most successful slimmer spending time with him and eating with him to the exclusion of the other three.

Discussion

The patients in this study were consecutive referrals from the same urban catchment area in south-west London. Only four patients (3.6%) described incest: a rate substantially less than those reported from other centres dealing with the eating disordered. Furthermore, two patients began their relationships as adults and thus, only 1.8% of this catchment area sample of bulimics reported childhood incest. Three patients reported unlawful sexual intercourse as a child and a further three had gross physical sexual abuse: a total of 5.4% (Table II). Statistical comparisons with the MORI and BBC surveys are not possible, but while the bulimia clinic figures are high, they are not too dissimilar from these general population surveys, especially as the methods used in this study were more exhaustive.

This study has taken a deliberately narrow view of childhood abuse. Verbal, and other more subtle, abuses outside the family and unencapsulated by fantasy have not been reported. Such abuse – broadly defined – of little girls seems common, difficult to quantify, variable in effect both at the time and subsequently. While not minimising or dismissing their effects on some, it is the victimised relationships described in this paper which, in bulimics at least, dominate subsequent therapy.

The author does not believe that CSA and incest cases in this study were missed. The enquiries were sensitively made at the end of a long interview whose aim, apart from data collection, was to establish a therapeutical alliance of honesty before treatment. Before sexual abuse was discussed, the patients had been giving, over a three-hour interview, intimate details of their family and sexual relationships, and of their eating disorder (which, in the author's long clinical experience dealing with this population, dominates their lives and about which they have more shame and guilt than any sexual matters). Before answering questions about abuse, the patients were instructed to pause so that they did not answer in a knee-jerk fashion. Lastly, and perhaps most importantly, virtually all patients went into subsequent psychotherapy with, in the main, female therapists and no further cases came to light.

An additional 18 patients reported incestuous fantasy. These were not transient recollections from adolescence but current preoccupations of importance which caused distress. Many patients reported uncomfortable looks or unnecessary physical closeness. The relationship between, and significance of, fantasy and reality has been argued over by therapists for decades and the discussion has an honoured place in the history of psychiatry. The analytic literature indicates that the relationship can become blurred to the point of being indistinguishable, particularly if the patient has incorporated such thoughts into neurotic conflict. The strength of this study is that the information was gained at the first initial assessment and the leading questions embedded in a wide-ranging psychiatric interview, rather than being retrieved during or after subsequent therapy when the answers might have become confused by transferential issues.

Incest was reported by only two patients (1.7%) who presented with bulimia alone; the other six patients all had concurrent alcohol or drug abuse (Table I). Multi-impulsive bulimics (Lacey & Evans, 1986) seem clinically different from the majority of bulimic patients, not only to the extent of their self-damaging behaviour, but also their underlying personalities have psychopathic or borderline features. It was initially claimed that incest had few negative effects on the child. However, the current literature documents quite convincing sequelae (Creighton, 1987). Mrazek & Kempe (1981) identified three categories of adverse long-term effects which include eating disorders, alcohol and drug abuse, sexual disinhibition and severe interpersonal difficulties. The author's subsequent work with multi-impulsive bulimics, outside this study, has confirmed his impression that incest or CSA are commonly reported by them. This study strongly suggests, however, that the prevalences of incest and indecency among the majority of normal-weight bulimics who do not abuse alcohol or drugs is far from high. It is perhaps less than described in other neurotic populations and only slightly higher than figures derived from general population surveys.

Appendix

Clinical details

Case number	*Details*
1	Father approached her (13 years) when she was having a bath. He fondled her breasts and told her she was too fat. Subsequently fondling occurred under threat of violence. Later he demanded fellatio: "it was softer that way – there was less violence; and afterwards he left me alone". Patient had epileptic fit (14 years) confirmed by electroencephalogram (EEG). "I was terrified of what would happen to me when I was unconscious". "I hated my body and what it was doing to our family". Guilt at presentation. Father has confirmed history of depression and alcohol abuse. Abuse 13–18 years.
31	Patient was aware that her maternal grandfather had molested her elder sister. Nonetheless, she allowed herself to be alone with him on a rowing boat (14 years). Kissing and fondling led to vaginal intercourse. She felt guilt. "I suppose I knew he might kiss me but never what happened". The relationship "was something quite horrid", but the event (which was repeated) became confused with her normal sexual feelings and her feelings for her father (sic). She began having sexual fantasies towards him also. She became frightened (17 years) when her father's new woman friend refused to live with him leaving the patient alone in the house with her father. Felt this "could lead to the same thing happening again". Binge-eating began in this context. Incest 13–16 years.
49	Fondled by father on a number of occasions (13–16 years). He penetrated her anus with his fingers. She described her manner towards him was precocious and 'flirting', "I could always get what I wanted". Received many presents from him. Later married an older man who subsequently left her. Drifted into a number of relationships with older married men (25 years her senior) who bought her expensive presents. Tendency to anal intercourse. Used suppositories as a means of weight control.
55	Father, described as 'selfish, puritan', had a history of alcohol abuse. Aware of him 'watching me'. Patient knew of father's incestuous relationship with her elder sister. "I felt there was no escape". "He was jealous of my boyfriend – he accused me of having sex with him; said I was disgusting". Fondling, masturbation but no intercourse (15–18 years).
75	At 17 years, patient regularly but briefly (six months) slept with her mother. Each fondled the other. Mutual masturbation. The episode occurred at the ending of her mother's extra-marital affair. Mother moved out of her husband's bed. "She was very upset, I went to console her". Very close to her father, "feelings for him also"; felt "I would end with one of them". Sexual disinhibition (12 partners in eight months).
88	Regular and gross abuse, including fellatio by father from six years until he left the family

continued

Appendix continued

when she was 12 years. Father alcoholic and violent with frequent psychiatric admissions. Prisoner of war. Patient angry towards her mother who was obese and "always heaped my plate, I suppose it was a reaction to the war". Father has confirmed history of depression with five hospital admissions.

94 Born in Guyana, illegitimate only daughter of a black African mother and an Indian father. Felt unwanted by her mother, claiming she was treated differently from her step-siblings. At 13 years she was violently raped by her stepfather and became pregnant. She was held responsible for this by her mother who arranged the abortion. Subsequently, intercourse with her stepfather continued and at times she was raped. Ran away from home five times. The patient's mother continued to declare that her husband was being seduced by the patient's precocious sexual behaviour and at 14 years she was taken into care. Abuse 13–14 years.

96 Only child of mother's second marriage which broke up when patient was six years. Father was distinguished Rhodes scholar, Triple Oxford Blue, in Colonial Service. 'Hard living', heavy drinker. Mother 'strong' and 'manipulative'. Patient did not see father after separation until she joined him on a holiday at 17 years. She was raped (with violence and under threat), but subsequently intercourse took place without threats. Patient had been sexually active from ten years and was 'promiscuous' after "my affair (sic) with my father" which was 'disgusting' but 'inevitable'. Later, patient discovered her step-sister had been seduced 11 years earlier and this had led to the parental divorce. Incest 17–19 years.

Acknowledgements

The author acknowledges with gratitude the anonymous help of a referee, Mr Peter Rooke, Barrister-at-Law, and Dr Nigel Eastman.

References

Abraham, S. F., Beumont, P. J. V. (1982) Varieties of psychosexual experience in patients with anorexia nervosa. *International Journal of Eating Disorders*, **1**, 10–19.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

Baker, A. W. & Duncan, S. P. (1985) Child sexual abuse: a study of prevalence in Great Britain. *Child Abuse Neglect*, **9**, 457–467

Bluglass, R. (1979) Incest. *British Journal of Hospital Medicine*, **22**, 152–157.

Calan, R. & Slade, P. (1987) Eating problems and sexual experience: some relationships. *British Review of Bulimia & Anorexia Nervosa*, **2**, 37–43.

Creighton, J. (1987) Child abuse in 1986. *Findings from the NSPCC's Register Research* (research briefing no. 8). London: National Society of Protection of Cruelty to Children.

Crisp, A. H. (1967) Anorexia nervosa. *Hospital Medicine*, **1**, 713–718.

—— (1984) The psychopathology of anorexia nervosa: getting the 'heat' out of the system. In *Eating and its Disorders* (eds A. J. Stunkard & E. Stellar). New York: Raven Press.

Finkelhor, D. (1979) *Sexually Victimised Children*. New York: Free Press.

Lacey, J. H. (1987) *'A Demographic and Clinical Study of Normal-Weight Bulimic Women in a London District Catchment Area'*. MD thesis, St Andrews/Dundee.

Lacey, J. H. & Evans, C. D. H. (1986) The impulsivist: a multi-impulsive personality disorder. *British Journal of Addiction*, **81**, 715–723.

Markowe, H. L. J. (1988) The frequency of childhood sexual abuse in the UK. *Health Trends*, **20**, 2–6.

Mrazek, P. J. & Kempe, H. (1981) *Sexually Abused Children and their Families*. Oxford: Pergamon Press.

——, Lynch, M. & Bentovim, A. (1983) Sexual abuse of children in the UK. *Child Abuse & Neglect*, **7**, 147–153.

Mullen, P. E., Romans-Clarkson, S., Walton, K., *et al* (1988) Effect of sexual and physical abuse on women's mental health. *Lancet*, **41**, 841–845.

Nash, C. L., West, D. J. (1985) Sexual molestation of young girls. In *Sexual Victimisation* (ed. D. J. West), pp. 1–92. Aldershot: Gower.

Oppenheimer, R., Howells, K., Palmer, R. L., *et al* (1985) Adverse sexual experiences in children and clinical eating disorders. *Journal of Psychiatry Research*, **19**, 357–361.

Schecter, M. D. & Roberge, L. (1976) Sexual exploitation. In *Child Abuse & Neglect: The Family and the Community* (eds R. E. Helfer & C. H. Kempe), pp. 127–142. Cambridge, Mass: Ballinger.

Sloan, G. & Leichner, P. (1986) Is there any relationship between sexual abuse or incest and eating disorders? *Canadian Journal of Psychiatry*, **31**, 656–660.

Wyatt, G. E. (1985) The sexual abuse of Afro-American & White American women in childhood. *Child Abuse & Neglect*, **9**, 507–519.

J. Hubert Lacey, MD, MPhil, FRCPsych, *Reader and Head, Adult Psychiatry Section, Academic Department of Psychiatry, St George's Hospital Medical School, Jenner Wing, Cranmer Terrace, London SW17 0RE*

British Journal of Psychiatry (1990), 157, 404–409

Hypochondriasis and Somatosensory Amplification

ARTHUR J. BARSKY and GRACE WYSHAK

A self-report questionnaire completed by 177 out-patients showed that hypochondriasis and amplification had a zero-order correlation of 0.56, and in stepwise multiple regression amplification accounted for 31% of the variance in hypochondriasis, after sociodemographic variables had been accounted for. Fears of ageing and death, and a childhood history of illness in the family, increased the R^2 to 0.50. Amplification was more powerful in women than in men and was also a significant (although weaker) correlate of somatisation, explaining 12% of the variance. Somatisation also correlated with being female, the propensity to seek medical care, and a diminished sense of efficacy over one's health. Our findings are consistent with the possibility that somatosensory amplification occurs in hypochondriasis.

While hypochondriasis has often been conceptualised from psychodynamic, interpersonal, and behavioural perspectives, it can also be understood as a disorder of perception and cognition (Barsky & Klerman, 1983). DSM–III–R (American Psychiatric Association, 1987) in fact emphasises the hypochondriac's faulty "interpretation of physical signs and sensations as evidence of physical illness". According to this model, hypochondriacs amplify benign somatic sensations and misattribute them to serious disease. Hypochondriacs, then, may experience a broad range of somatic sensations as more intense, more noxious, more ominous, and more disturbing than non-hypochondriacs. They may be unusually sensitive to, and intolerant of, bodily sensations in general, and be more likely to attribute them to disease.

The hypochondriac may amplify a wide range of sensations: normal physiological functions; the benign symptoms of trivial and self-limited ailments; the somatic concomitants of emotional arousal; as well as the symptoms of serious disease. He or she may thus be characterised as having an amplifying perceptual style entailing a hypervigilance or heightened attentional focus upon bodily sensation, a tendency to select out and attend to certain relatively weak and infrequent bodily sensations, and a propensity for responding to them with affect and cognitions which make them more disturbing and intense.

Because his or her bodily sensations are so intense and so disturbing, the hypochondriac then tends to misinterpret them and incorrectly attribute them to a serious disease rather than to a more benign cause such as overwork, insufficient sleep, inadequate exercise, etc. Thus, for example, a normal irregularity in the consistency of breast tissue is mistaken for a 'lump', or a stomach-ache is attributed to an ulcer rather than to a recent dietary indiscretion. Having hypothesised that he or she is sick, the hypochondriac becomes increasingly apprehensive and hypervigilant. This heightens somatic symptoms further, via two mechanisms. Firstly, people screen their somatic perceptions and selectively attend to those that confirm their explanatory hypotheses, while ignoring sensory input that disconfirms their beliefs (Pennebaker, 1982). Secondly, the individual's mounting anxiety produces its own set of somatic symptoms, which are actually benign, but are incorrectly ascribed to the presumed serious disease. The result is a further heightening of alarm, and a vicious cycle ensues.

In this conceptualisation of hypochondriasis, the other clinical characteristics of the disorder are viewed as secondary phenomena. Given the hypochondriac's somatic experience, he or she quite understandably becomes obsessed with health and preoccupied with his or her body. Given the doctor's inability to explain or treat his or her symptoms, the hypochondriac naturally becomes dissatisfied, feels antagonistic and angry about his or her predicament, and makes multiple medical visits. The hypochondriacal characteristics are, in short, the understandable consequence of the patient's disordered perceptions and cognitions.

Several findings support this conceptualisation of hypochondriasis. Firstly, college students who are more hypochondriacal have a higher level of sensory arousal and are more sensitive to paired flashes of light (Hanback & Revelle, 1978). Secondly, normal subjects who are kinaesthetic augmenters (overestimating the size of objects placed in their hands while blindfolded), score highest on hypochondriasis scales and have a lower tolerance for experimental pain (Petrie, 1978). In work with patients who had chronic, organic, non-malignant pain, those with higher hypochondriasis scores rated their pain as more intense (Ziesat, 1978). Among patients with pain complaints, those with disease conviction and disease phobia have lower thresholds and tolerance to experimental pain (Merskey & Evans, 1975), and psychiatric in-patients with disease phobias were also found to have lower thresholds for, and tolerance of, experimental pain (Bianchi, 1971, 1973). However, there is, on the contrary, at least one study (Miller *et al*, 1981) which failed to find a relationship between hypochondriasis and a heightened awareness of internal bodily sensation – 'private body consciousness'.

Two studies suggest that hypochondriacs are more sensitive to normal physiological sensations. In one, workers who reported respiratory symptoms disproportionate to their pulmonary function tests were found to be more hypochondriacal (Wright *et al*, 1977). Furthermore, among psychiatric out-patients, those who were hypochondriacal were more aware of their cardiac function, determined by actual pulse rate, than those who were phobic (Tyrer *et al*, 1980).

There is also a literature, primarily in experimental and cognitive psychology rather than in clinical medicine, indicating that the cognitive processing of sensation can amplify it: the thoughts, opinions, and beliefs a person holds about his or her physical state powerfully affect his or her level of discomfort. Thus, the experimental manipulation of subjects' beliefs about the causes of their sensations has been shown to reduce distress and arousal (Rodin, 1978; Valins & Nisbett, 1971). Furthermore, misattributions of disease can result in increased symptoms: when apparently healthy individuals receive screening chest X-rays, 8% of those told that the X-ray revealed possible cardiac disease developed new symptoms referable to the heart, although their cardiac status did not change (Wheeler *et al*, 1958). In a more clinical vein, the health concerns of subjects who scored highest on a hypochondriasis scale have been judged to result from the misattribution of bodily sensations (Rodin, 1978).

In previous work (Barsky *et al*, 1986*a*), we found that four core hypochondriacal symptoms (disease fear, disease conviction, bodily preoccupation, and somatic symptoms) tend to cluster together in some ambulatory medical patients in a way consistent with the DSM–III diagnosis. In the present study, we sought evidence that the misinterpretation of benign physical signs and sensations as indicative of serious disease, also included in the DSM–III description, occurs in these patients. The study employed a self-report questionnaire measuring somatosensory amplification to determine whether amplification was related to hypochondriasis and to somatisation. Because of the preliminary nature of this work, it was clear from the outset that while this study could generate hypotheses, it would be unable to test them definitively.

Method

The study was conducted in the general medical out-patient clinic of the Massachusetts General Hospital. The clinic provides primary care to 29 000 patients, accounting for over 50 000 visits annually. It is staffed by 36 physicians and 65 house officers.

The study sample consisted of consecutive patients attending the clinic on randomly chosen days. Fluency in English and the patient's having been followed in the clinic for at least one year prior to the index visit were necessary for inclusion in the survey. The only exclusion criterion was severe organic brain disease. Of 230 patients asked to participate, 185 consented, and complete data were obtained on 177.

Subjects, aided by a research assistant, completed a 93-item self-report questionnaire while at the clinic. A small private area was provided for the purpose. This assured confidentiality and minimised the possibility that subjects seated near each other would discuss their responses.

The battery contained a questionnaire about sociodemographic characteristics; self-report scales measuring hypochondriasis, amplification, and somatisation; and a questionnaire to assess several attitudes toward health and medical care, beliefs about illness, and health practices. All scales were constructed with a five-point Likert response format from one ('strongly agree' or 'very true of me') to five ('strongly disagree' or 'not at all true of me').

As no 'gold standard' exists for the diagnosis of DSM–III–R hypochondriasis, the hypochondriacal syndrome was assessed with the Whiteley Index, a 13-item questionnaire developed by Pilowsky (1967) which focuses upon hypochondriacal attitudes and beliefs. On principal-components analysis, it yields three factors: disease conviction, disease fear, and bodily preoccupation. Its test–retest reliability has been established, it is valid in discriminating psychiatric patients diagnosed as hypochondriacal from those who are not, and there is a high concordance between the Whiteley score and spouses' ratings of hypochondriasis (Pilowsky, 1967). Whiteley Index scores are associated with several clinical characteristics thought to be secondary features of hypochondriasis, such as excessive use of medical care and complaints that are disproportionate to demonstrable pathology (Hanback & Revelle, 1978; Ziesat, 1978; Pilowsky & Spence, 1977; Beaber & Rodney, 1984; Kasteler *et al*, 1976; Wright *et al*, 1977).

Somatisation, generally defined as the tendency to express emotional dysphoria as somatic symptoms, was assessed with 11 somatic symptoms drawn from the Hopkins Symptom Check-list somatisation subscale (Derogatis *et al*, 1974, 1981), and the Minnesota Multiphasic Personality Inventory (MMPI) hypochondriasis subscale. Typical symptoms queried include backache, itching, 'upset stomach', palpitations, numbness, and fatigue. While any of these symptoms can be caused by a serious medical disorder, they are also common 'functional' complaints. Prior work with this scale has disclosed a test–retest reliability of 0.86 in 75 patients over an interval of 1–5 weeks.

Amplification was measured with a five-item self-report instrument which asks about the respondent's sensitivity to a range of normal bodily sensations ("I am quick to sense the hunger contractions of my stomach" and "I'm often aware of various things happening in my body") and to neutral and noxious stimuli ("Loud noises really bother me", "I am very aware of changes in my body temperature", and "I cannot stand pain as well as most people can"). These items, scored on the five-point Likert scale, were selected from an item pool composed of unpleasant and uncomfortable sensations volunteered by medical outpatients. A similar item on bodily awareness was eliminated from the Whiteley Index to obviate the problem of covariance between the two scales. Two others are similar to items in Miller's Body Consciousness Questionnaire (Miller *et al*, 1981). Its psychometric properties are the subject of a separate report; in this study, a ten-item version of the scale had a test–retest reliability of 0.85 (coefficient of reproducibility) when administered to 52 medical outpatients over an interval of 28 to 146 days (mean = 72 days), and an internal consistency of 0.70 (Cronbach's alpha).

Attitudes to health, illness and medical care were assessed on a 50-item questionnaire focusing on fears of ageing and death, a feeling of somatic vulnerability to illness and injury, the value placed on health, the propensity to seek medical care, a sense of personal efficacy over one's own health, and a childhood history of illness in the family. These items have face validity and were selected in preliminary work from a 91-item pool composed of common patient responses to questions about each of these areas. This approach was adopted because scales of established reliability and validity do not exist to assess most of these attitudes.

Product moment correlations were performed to determine the association between two variables. Stepwise multiple regression was done to determine the proportion of the variance of a dependent variable accounted for by independent or predictor variables. Whenever a variable enters the regression equation, its incremental contribution to the variance is independent of the variables that have entered in preceding steps.

Results

The mean age of the sample was 54.2 years (s.d. 17.4, range 18–86). Sixty-two per cent (109 patients) were women and 92% were white. Religious affiliation was given as 61% Catholic, 23% Protestant, 6% Jewish, and 10% other. With regard to marital status, 54% were married, 33% were single, divorced or separated, and 13% were widowed. A total of 14% were in social classes I and II, 31% in class III, 27% in class IV, and 28% in class V (Hollingshead & Redlich's (1958) two-factor index of social position).

We first examined the psychometric properties of the instruments used. The intra-scale consistency of the 13-item Whiteley Index was 0.84 (Cronbach's alpha) and that of the 11-item somatisation scale was 0.79. The amplification scale, in part because it was composed of only five items, had a Cronbach's alpha of 0.45. The Cronbach's alpha values of the other subscales were as follows: the propensity to seek medical care (four items) = 0.66; the value placed on health (four items) = 0.53; fear of ageing and death (three items) = 0.43; somatic vulnerability (three items) = 0.36; and sense of efficacy over health (three items) = 0.30.

We then studied the relationship between hypochondriasis and amplification. Their zero-order correlation coefficient was 0.56 ($P = 0.0001$). A stepwise multiple regression, employing hypochondriasis as the dependent variable and including all the study patients, is presented in Table I. The demographic variables, age, sex, and married/not married were forced into the regression model. The stepwise procedure then entered variables in the order of their contribution to the total variance. None of the three demographic variables was significantly associated with hypochondriasis ($R^2 =$ 0.6%). Amplification is the single most powerful predictor of hypochondriasis, increasing R^2 from 0.6% to 31%, followed by fear of ageing and death, and childhood history of illness which raised the R^2 to 50%. The interaction term, sex × married/not married, was significant and further increased R^2 to 53%. The coefficient of this term is negative, indicating a differential effect of marriage on men and women. With the inclusion of the interaction term, sex and married/not married become statistically significant. The next independent variable to enter the equation was the value placed on health, but it did not increase R^2 significantly. Other variables which did not contribute significantly were attitude toward utilisation and the interaction terms sex × childhood history and sex × amplification.

When we examine men and women separately (Table II), the differential influence of married/not married is apparent – among men, not being married (single, widowed or divorced) is positively associated with hypochondriasis, while among women being married is related to hypochondriasis. Further, amplification is somewhat more powerful in women, accounting for 34% of the variance in hypochondriasis.

Separate regression analyses of three of the principal symptoms of DSM-III-R hypochondriasis (there is a Whiteley Index subscale for each) reveal that amplification plays a lesser role in bodily preoccupation than in disease fear or disease conviction (Table III) while fear of ageing and death appears more closely related to bodily preoccupation than to the other two symptoms

We also examined the relationship between amplification and somatisation. The zero-order correlation between hypochondriasis and somatisation was 0.58 ($P < 0.0001$), and between amplification and somatisation was 0.44 ($P < 0.0001$). The results of a stepwise regression against

TABLE I

Correlates of hypochondriasis, in multiple stepwise regression (n = 177)

Step	*Variable*	b[1]	R^2	P[1]
0	Age	0.006		0.78
	Sex	0.39	0.006	0.0027
	Marital status	0.34		0.012
4	Amplification	0.41	0.31	0.0001
5	Fear of ageing/death	0.29	0.45	0.0001
6	Childhood history of illness	0.13	0.50	0.0001
7	Sex × marital status	−0.56	0.53	0.001

1. *P* and *b* values are those obtained at the last step.

TABLE II
Correlates of hypochondriasis by sex, in multiple stepwise regression (n = 177)

Men (n = 68)					*Women (n = 109)*				
Step	*Variable*	b	R^2	P	*Step*	*Variable*	b	R^2	P
1	Fear of ageing/death	0.26	0.28	0.0003	1	Amplification	0.43	0.34	0.0001
2	Amplification	0.31	0.44	0.0017	2	Fear of ageing/death	0.30	0.45	0.0001
3	Childhood history	0.18	0.49	0.0018	3	Childhood history	0.11	0.50	0.0016
4	Marital status	0.39	0.55	0.0059	4	Marital status	−0.20	0.52	0.04

TABLE III
Correlates of hypochondriacal symptoms in multiple stepwise regression (n = 172[1])

Step	*Variable*	b	R^2	P
Disease conviction				
1	Amplification	0.31	0.16	0.0001
2	Childhood history	0.17	0.25	0.0001
3	Fear of ageing/death	0.25	0.32	0.0003
Disease fear				
1	Amplification	0.33	0.18	0.0001
2	Fear of ageing/death	0.23	0.26	0.0003
3	Age	0.01	0.28	0.0054
4	Sex	0.30	0.30	0.0059
5	Childhood history	0.08	0.32	0.0421
Bodily preoccupation				
1	Fear of ageing/death	0.43	0.14	0.0001
2	Childhood history	0.18	0.19	0.0056
3	Amplification	0.34	0.23	0.0062
4	Sex	−0.38	0.24	0.0372

[1] Program could not analyse data for 5 of the 177 subjects.

TABLE IV
Correlates of somatisation in multiple stepwise regression

Step	*Variable*	b[1]	R^2	P[1]
0	Age	0.01		0.0014
	Sex	−0.89	0.11	0.0001
	Marital status	−0.17		0.0662
4	Propensity to seek medical care	0.32	0.31	0.0001
5	Amplification	0.30	0.43	0.0001
6	Sense of efficacy	0.26	0.48	0.0001
7	Sex × childhood history	0.13	0.51	0.0005
8	Fear of ageing/death	0.12	0.53	0.0118

[1] *b* and *P* values are those obtained at the last step.

somatisation are presented in Table IV. Age, sex and married/not married were forced into the equation; only sex was significantly associated with somatisation – the coefficient is negative, indicating that female gender is associated with somatisation. The R^2 is 11%. The propensity to seek medical care raises R^2 to 31%; with the inclusion of this variable, age becomes a significant predictor of somatisation, indicating that when the tendency to seek medical care is controlled for, age and somatisation are positively correlated. Amplification enters next, raising R^2 to 43%, followed by a diminished sense of efficacy over health. The interaction term, sex × childhood history of illness, is significant with a positive coefficient, indicating an association in women, but not in men, which further raises R^2 to 51%. A small but significant incremental contribution is made by fears of ageing and death. Variables eligible for inclusion which failed to meet the criterion for inclusion ($P \leq 0.05$) were: the interaction term, sex × greater value placed on health (more marked among women than men), physical fitness, and the interaction terms sex × amplification and sex × married/not married.

Discussion

This work suggests that there is a relationship between amplification, as measured by a brief self-report questionnaire, and hypochondriacal attitudes; and between amplification and the bodily complaints characteristic of somatisation. Amplification explains 31% of the variance in hypochondriasis and 12% of the variance in somatisation, after sociodemographic descriptors have been taken into account. Since hypochondriasis was assessed only with a questionnaire, however, these findings cannot necessarily be generalised to DSM-III-R hypochondriasis.

The term hypochondriasis has at least two different meanings. DSM-III-R refers to a distinct and discrete disorder, a primary psychopathological condition which is found in some individuals but not in most. It is thought to be a primary disorder because these symptoms tend to occur together, without other underlying psychiatric disorders of which they are a secondary feature, and to exhibit a characteristic clinical presentation, course, and response to treatment. Conviction of disease, fear of disease, and bodily preoccupation are central features of the DSM-III-R criteria as well as the hypochondriac's misattribution of non-pathological signs and sensations to serious disease. In contrast, hypochondriasis has also been viewed as a non-specific collection of symptoms (a syndrome) which are distributed more continuously throughout a population and which are secondary features of other more pervasive psychiatric disorders (Kenyon, 1964). In this sense, hypochondriasis is analogous to dyspnoea or jaundice rather than to asthma or hepatitis.

In this paper we have discussed hypochondriasis as a primary disorder. But no criterion standard ('gold standard') exists with which to make that diagnosis. We have therefore employed a self-report questionnaire which measures the degree to which the component symptoms of hypochondriasis are present, but which does not provide a clinical diagnosis. Strictly speaking, then, our data only concern a relationship between amplification and hypochondriacal symptoms, not a relationship between amplification and DSM-III-R hypochondriasis. The two definitions of hypochondriasis, however, appear to be closely related. We previously found (Barsky *et al*, 1986*a*,*b*) that the component symptoms of hypochondriasis, assessed with the Whiteley Index, do tend to cluster together in a subset of ambulatory medical patients in a way which appears compatible with DSM-III-R hypochondriasis (Barsky *et al*, 1986*a*,*b*). To the degree that the Whiteley Index corresponds to DSM-III-R hypochondriasis, our data support the validity of DSM-III-R's emphasis upon the misattribution of non-pathological signs and sensations to serious disease. Our findings suggest that hypochondriacs are disturbed not only by the pathological symptoms of disease, but also by bodily sensations which do not suggest disease; the amplifier notices and is bothered by a range of noxious sensations that are not pathological *per se*, such as noise, heat, and hunger. The amplification questionnaire includes items about hunger and pain, and our results are therefore consistent with previous findings that hypochondriacs are more sensitive to some normal physiological sensations, and that they are less tolerant of experimental pain (Petrie, 1978; Tyrer *et al*, 1980; Wright *et al*, 1977.)

These data do not necessarily imply a causal relationship between amplification and hypochondriasis. Since the study is cross-sectional rather than longitudinal, it is not possible to determine the direction of causality. It is as possible that hypochondriacal concerns cause people to amplify bodily sensations as it is that an amplifying style leads to hypochondriasis. Another caution in interpreting the results stems from the nature of the sample. This is a medical population, and the relationship between hypochondriasis and amplification could be confounded by medical morbidity, a variable we did not assess. Zonderman *et al* (1985) pointed out the danger of concluding that a patient's fears and concerns about disease are hypochondriacal without assessing his or her medical status at the same time. It is possible, for example, that medical morbidity itself causes both hypochondriacal symptoms and amplification. Our previous work, however, suggests that hypochondriacal symptoms, as measured by the Whiteley Index, are not significantly related to the number of major medical diagnoses that a patient carries (Barsky *et al*, 1986*a*). A third caveat in interpreting these findings stems from the possible effect of other concurrent psychiatric morbidity (particularly depressive and anxiety disorders) which is prevalent in hypochondriasis, and was not assessed in this study. It is possible that amplification is a function of these other disorders, or even of psychiatric disorder in general, rather than being more specifically related to hypochondriasis. Further work is necessary to elucidate these relationships.

Amplification appears to be somewhat more important in women, and less closely related to bodily preoccupation than to the fear and conviction of disease. The gender difference is compatible with many reports that women report more somatic symptoms and more minor symptoms than men do (Pennebaker, 1982). We did not, however, find amplification directly related to gender: the zero-order correlation between sex and amplification was not statistically significant. Furthermore, no previous work suggests that female hypochondriacs have a markedly different clinical presentation from that of males.

The regression equations also point to a relationship between hypochondriasis and both fears of death and childhood experiences of illness ("a lot of illness in my family when I was growing up"). Both of these findings are consistent with previous work. Kellner (1986), for example, reported a heightened fear of death and dying among hypochondriacs. A childhood history of illness was measured with only a single item in this study, and it is a variable that is highly susceptible to retrospective falsification: the hypochondriac's frequent illness experience in adulthood may colour his or her memory of childhood. One prospective study, however, has linked childhood experiences to subsequent amplification, finding an association between maternal over-attentiveness to benign illness in childhood and bodily hypervigilance in adulthood (Mechanic, 1980). This is also consistent with the general impression in the literature that during their childhoods, hypochondriacs are frequently exposed to medical illnesses and functional symptoms in family members (Kellner, 1986; Barsky, 1983, 1988).

The data also suggest that amplification may be related to the more general process of somatisation. Amplification explained a modest but significant proportion of the variance in somatisation; therefore, it may not be a unique correlate of hypochondriasis that distinguishes it from somatisation in general. However, it must be reiterated that this is a medical population, and we did not assess the degree to which these bodily complaints reflect medical disease rather than somatised dysphoria.

The concept of amplification deserves further investigation, particularly in studies which control for medical morbidity and other concurrent psychopathology. It is important to demonstrate the convergent validity of the self-report questionnaire with perceptual tests such as cortical evoked potentials. In addition, the state versus trait nature of amplification deserves further investigation. Once the concept is on firmer ground, then further studies of its clinical role in somatoform disorders and in somatised depression and anxiety disorders would be in order.

Acknowledgement

This investigation was supported in part by research grant MH 40487 from the National Institute of Mental Health.

References

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

Barsky, A. J. (1988) Somatoform disorders. In *Comprehensive Textbook of Psychiatry* (5th edn) (eds. H. I. Kaplan & B. J. Sadock). Baltimore: Williams & Wilkins.

—— & Klerman, G. L. (1983) Overview: hypochondriasis, bodily complaints, and somatic styles. *American Journal of Psychiatry*, **140**, 273–283.

——, Wyshak, G. & Klerman, G. L. (1986*a*) Hypochondriasis: an evaluation of the DSM-III criteria in medical out-patients. *Archives of General Psychiatry*, **43**, 493–500.

——, —— & —— (1986*b*) Medical and psychiatric determinants of out-patient medical utilization. *Medical Care*, **24**, 548–560.

Beaber, R. J. & Rodney, W. M. (1984) Underdiagnosis of hypochondriasis in family practice. *Psychosomatics*, **24**, 39–45.

Bianchi, G. N. (1971) Origins of disease phobia. *Australian & New Zealand Journal of Psychiatry*, **5**, 241–257.

Bianchi, G. N. (1973) Patterns of hypochondriasis: a principal components analyses. *British Journal of Psychiatry*, **122**, 541–548

Derogatis, L. R., Lipman, R. S., Rickels, K., *et al* (1974) The Hopkins symptom checklist (HSCL): a self-report symptom inventory. *Behavioral Science*, **19**, 1–15.

Derogatis, L. R., Rickels, K., Uhlenhuth, E. H., *et al* (1981) The Hopkins symptom checklist. In *Psychological Measurements in Psychopharmacology* (eds P. Pichot & R. Oliver-Martin), pp. 79–110. Basel: Karger.

Hanback, J. W. & Revelle, W. (1978) Arousal and perceptual sensitivity in hypochondriasis. *Journal of Abnormal Psychology*, **87**, 523–530.

Hollingshead, A. B. & Redlich, F. C. (1958) *Social Class & Mental Illness: A Community Study*. New York: Wiley

Kasteler, J., Kane, R. L., Olsen, D. M., *et al* (1967) Issues underlying prevalence of "doctor-shopping" behavior. *Journal of Health and Social Behavior*, **17**, 328–339.

Kellner, R. (1986) *Somatization and Hypochondriasis*. New York: Praeger.

Kenyon, F. E. (1964) Hypochondriasis: a clinical study. *British Journal of Psychiatry*, **100**, 478–488.

Mechanic, D. (1980) The experience and reporting of common physical complaints. *Journal of Health and Social Behavior*, **21**, 146–155.

Merskey, H. & Evans, P. R. (1975) Variations in pain complaint threshold in psychiatric and neurological patients with pain. *Pain*, **1**, 73–79.

Miller, L. C., Murphy, R. & Buss, A. H. (1981) Consciousness of body: private and public. *Journal of Personality and Social Psychology*, **41**, 397–406.

Pennebaker, J. W. (1982) *The Psychology of Physical Symptoms*. New York: Springer-Verlag.

Petrie, A. (1978) *Individuality in Pain and Suffering* (2nd edn). Chicago: University of Chicago Press.

Pilowsky, I. (1967) Dimensions of hypochondriasis. *British Journal of Psychiatry*, **113**, 89–93.

Pilowsky, I. & Spence, N. D. (1977) Ethnicity and illness behavior. *Psychological Medicine*, **7**, 447–452.

Rodin, J. (1978) Somatopsychics and attribution. *Personality and Social Psychology Bulletin*, **4**, 531–540.

Tyrer, P , Lee, I. & Alexander, J. (1980) Awareness of cardiac function in anxious, phobic and hypochondriacal patients. *Psychological Medicine*, **10**, 171–174.

Valins, S. & Nisbett, R. E. (1971) Attribution processes in the development and treatment of emotional disorder. In *Attribution: Perceiving the Causes of Behavior* (eds E. E. Jones, D. K. Karouse & H. H. Kelley). Morristown, NJ: General Learning.

Wheeler, E. O., Williamson, C. R. & Cohen, M. E. (1958) Heart scare, heart surveys, and iatrogenic heart disease. *Journal of the American Medical Association*, **167**, 1096–1102.

Wright, D. D., Kane, R. L., Olsen, D. M., *et al* (1977) The effects of selected psychosocial factors on the self-reporting of pulmonary symptoms. *Journal of Chronic Disease*, **30**, 195–206.

Ziesat, H. A. (1978) Correlates of the tourniquet ischemia pain ratio. *Perceptual and Motor Skills*, **47**, 147-150.

Zonderman, A. B., Heft, M. W. & Costa, P. T. (1985) Does the Illness Behavior Questionnaire measure abnormal illness behavior? *Health Psychology*, **4**, 425–436.

*Arthur J. Barsky, MD, *Department of Psychiatry, Harvard Medical School, and Psychiatry Service and Primary Care Program, Massachusetts General Hospital, ACC 807, Fruit Street, Boston, MA 02114, USA;* Grace Wyshak, PhD, *Department of Medicine, Harvard Medical School and Harvard Center for Population Studies, Harvard School of Public Health, 9 Bow Street, Cambridge, MA 02138*

*Correspondence

British Journal of Psychiatry (1990), **157**, 410–415

Are British Psychiatrists Racist?

GLYN LEWIS, CAROLINE CROFT-JEFFREYS, ANTHONY DAVID

Out of a sample of 220 British psychiatrists, 139 completed a questionnaire regarding a case vignette of a psychotic illness. The sex and 'race' of the vignette were varied and the responses compared. The Afro-Caribbean case was regarded as that of an illness of shorter duration, and requiring less neuroleptics than the white case. Respondents judged the Afro-Caribbean case as potentially more violent and thought criminal proceedings were more appropriate. The female vignette was perceived as less violent, less criminal, and less likely to need neuroleptics. Cannabis psychosis and acute reactive psychosis tended to be diagnosed more often and schizophrenia less often in Afro-Caribbean cases, refuting the claim that psychiatrists tend to overdiagnose schizophrenia in this group. Such 'race thinking' (a form of stereotyping which is distinct from ideological racism) could lead to inappropriate management.

'Race' is "a socially constructed categorisation which specifies rules for the identification of members. Racism is the application of 'race' categories in social contexts" (Husband, 1982). Fernando (1986) provides a more elaborate definition of racism: "behaviour that arises from a belief that people can be differentiated from one another mainly or entirely on the basis of their ancestral lineage . . . some physical characteristic, . . . sometimes by a behavioural characteristic, and that groups of people so characterised are to be treated as different . . . with one or more groups being inferior to others." This definition introduces the idea of inferiority as an integral part of racism. The difficulty surrounding this area has led to the distinction between ideological racism and everyday beliefs about 'race', sometimes called 'race-thinking' (or 'common-sense racism') (Husband, 1982). Race-thinking wrongly assumes that 'race' is an objective fact which explains differences between people, rather than an artificial, socially constructed label. However, unlike ideological racism, race-thinking does not refer to any sociobiological theory. Furthermore, the difficulties in defining inferiority, a debatable value judgement inherent in ideological racism, are avoided.

In the last decade there has been concern over the apparent increase in the rates of compulsory treatment, police involvement, and prescription of long-acting neuroleptics among Afro-Caribbean immigrants and their descendants with psychotic illness (Littlewood & Cross, 1980; Ineichen *et al*, 1984; McGovern & Cope, 1987; Fahy *et al*, 1987). One explanation for these worrying statistics is that psychiatrists and the police are not exempt from the racist beliefs and attitudes prevalent in British society (Burke, 1984), and that these attitudes could bias psychiatric diagnosis and treatment. Littlewood & Lipsedge (1981) suggested that psychotic reactions in British Afro-Caribbeans are more likely to be misdiagnosed as schizophrenia. In addition, Littlewood (1988) noted that the diagnosis of cannabis psychosis is applied more frequently in this population despite the lack of convincing evidence supporting a major aetiological role for cannabis in severe psychosis (Onyango, 1986). In short, it has been argued that mentally ill British Afro-Caribbeans are treated more punitively and more likely to be 'labelled' with the stigmatising diagnoses of schizophrenia or cannabis psychosis because of racist attitudes among psychiatrists (Littlewood & Lipsedge, 1981; Mercer, 1984; Cox, 1986; Littlewood, 1988).

Evidence has also emerged suggesting an increased incidence of schizophrenia among British Afro-Caribbeans (Rwegellera, 1977; Dean *et al*, 1981; Cochrane & Bal, 1989), despite using standardised assessment intended to minimise diagnostic bias (Harrison *et al*, 1988). This increased incidence has been used to explain differences in the rates of compulsory treatment (McGovern & Cope, 1987; Fahy, 1989), as such treatment and the use of biological therapies are appropriate given a diagnosis of schizophrenia. Some authors still implicate societal racism as a possible cause of the increased risk of schizophrenia (Littlewood & Lipsedge, 1988).

The purpose of the present study was to examine race-thinking in psychiatrists working in Britain. One approach to a psychological understanding of this phenomenon is the concept of stereotypes: "oversimplified, rigid and generalised beliefs about groups of people in which all individuals from the group are labelled with the perceived characteristics of the group" (Harré & Lambe, 1983). Stereotypes can be studied using the case-vignette method, in which subjects are randomly allocated short case descriptions and asked to complete questions on management, clinical features, and attitudes (e.g.

Lewis & Appleby, 1988). The information presented is kept sparse deliberately, to force the respondents to call upon their beliefs, attitudes, and prejudices; stereotypes are needed to complete the questionnaire. The experimental nature of this method is another important advantage. Critical variables in the 'cases' can be manipulated independently of all potential confounders and the respondents can be randomly allocated to the different conditions.

The case-vignette method has been used to study diagnostic bias according to 'race'. Loring & Powell (1988) in a sample of US psychiatrists found that diagnosis, using DSM-III (American Psychiatric Association, 1980), was influenced by the 'race' (and sex) of case vignettes, despite warnings that the reliability of their diagnoses would be examined. Ford & Widiger's (1989) study, using the same methodology, showed a sex bias in the diagnosis of DSM-III histrionic and antisocial personality disorders.

The attitudes, proposed management, and diagnoses of practising psychiatrists were studied using the case-vignette method. The aim was to examine how psychiatrists' responses differed when presented with Afro-Caribbean and white psychotic 'patients'.

Method

The names of 220 psychiatrists who lived in England, Wales or Scotland were randomly selected from the 1985 Membership List of the Royal College of Psychiatrists (approximately 10% of total – Department of Health and Social Security (1987)). Those who were described as registrars and senior registrars, those who were retired and those listed as being child psychiatrists were excluded. The survey was carried out in early 1989. Subjects were randomly allocated one of four brief case histories, which they were asked to read before completing and returning an accompanying questionnaire. They were told that we were interested in attitudes to community care. The real purpose of the study was not explained.

The four case histories differed from each other in one particular detail. The amount of information was deliberately restricted. The first case history was as follows.

> A 23-year-old woman has recently become unemployed. Her family called the police because she had damaged furniture, been acting in a threatening manner, and been verbally aggressive. She has no past psychiatric history but the family noted that she had spent more time in her room alone, reading religious books, which seemed to follow the death of a close friend. They also suspect that she had been smoking cannabis. Her father is a British Rail ticket clerk and her mother a housewife. There are two younger sisters and an elder brother. The only family history of mental illness is in an aunt who spent several years in a mental hospital. She drinks two units of alcohol per week and denies drug abuse.
>
> She is a fit white woman who appears agitated and paces around. She does not complain of depressed mood but says she heard a voice telling her that the world was ending. She also believes that the neighbours are plotting to kill her and that they are poisoning the milk. There are no other mental-state abnormalities.

This case was modified slightly in the following ways: case 2, the patient is an Afro-Caribbean female; case 3, the patient is a white male; case 4, the patient is an Afro-Caribbean male.

Subjects were asked to give their sex and age and provide details about previous experience, medical school of graduation, and main clinical setting (community, mental hospital, or general hospital). They were asked to record their views of mental illness on a biological–social continuum and their position on community care.

The questionnaire also included 23 semantic differentials with a six-point scale, designed to elicit one aspect of the assessment or management of the case. Most of the items placed emphasis on management and included statements designed to examine the hypothesis that racist attitudes would lead to a more custodial, less psychotherapeutic, less sympathetic plan of management (Table I). The semantic differentials were scored so that a higher score represented agreement with the statements listed in Table I. For instance, a response at the end of the scale 'Risk of violence to staff' scored five, and a response at the end 'No risk of violence to staff' was scored zero. It is difficult to know how the absolute values of the semantic differential scores would correspond to actual attitudes or practice. For this reason the analysis will concentrate on the *relative* differences between groups.

Each subject was asked to complete the questionnaire and then choose the three most likely diagnoses in order of preference from the following: schizophrenia, affective psychosis, cannabis psychosis, acute reactive psychosis, schizotypal personality disorder, and pathological grief reaction. The diagnoses were restricted in this way to simplify the analysis.

Results

Twenty-nine of the 220 psychiatrists were either retired or deceased. Of the remaining 191, 139 (73%) completed the questionnaire; a further 10 replied refusing to participate. There were 118 (85%) men and 21 (15%) women in the final sample. Mean age was 49 years (s.d. 9) and mean years of experience in psychiatry was 21 years (s.d. 7). There were 107 (79%) UK graduates, 16 (12%) had graduated from medical schools in less developed countries, and 13 (9%) graduated from other foreign medical schools (the other three had data missing). Sixty-three (47%) of the final sample worked mainly in mental hospitals, 43 (32%) in general hospitals, and 9 (7%) in the community. The remaining 19 (14%) for whom data was available worked in other settings, including the private sector.

There was no significant difference between the characteristics of the respondents on the four versions of the case vignette on age, medical school, or years of experience. However, there was a systematic distribution of sex of respondent ($\chi^2 = 9.2$, d.f. 3; $P = 0.03$). This is accounted for by the lack of female psychiatrists examining case 2 (black female), which occurred by chance, despite a correct randomisation procedure. Hence analyses which include the sex of respondent are not presented. We

TABLE I

Means (s.e.m.) scores for semantic differentials[1], by race of vignette

Statement about patient	*Race*		*Significance*		
			parametric:		*non-parametric:*
	white	*black*	*t*	P	*Wilcoxon*
Admission as an in-patient is indicated	3.79 (0.14)	3.68 (0.16)	0.53	0.60	0.60
Admission as a day patient is indicated	2.72 (0.19)	3.27 (0.22)	1.87	0.06	0.07
Section 2 indicated	3.01 (0.20)	3.13 (0.21)	0.39	0.69	0.70
Not a suicide risk	2.81 (0.15)	3.00 (0.16)	0.85	0.40	0.33
Risk of violence to staff	2.41 (0.15)	2.87 (0.13)	2.29	0.02[2]	0.03[2]
Risk of violence to family	2.86 (0.14)	3.08 (0.12)	1.12	0.27	0.39
Suitable for proposed community treatment order	2.62 (0.19)	2.69 (0.21)	0.24	0.81	0.85
Neuroleptic treatment indicated	4.26 (0.12)	3.71 (0.18)	2.57	0.01[2]	0.02[2]
Duration of illness likely to exceed three months	2.61 (0.20)	1.94 (0.19)	2.36	0.02[2]	0.03[2]
Cannot be managed in the community	2.92 (0.18)	2.77 (0.18)	0.57	0.57	0.53
Cannabis a major contributor to mental state	1.96 (0.15)	2.13 (0.16)	0.78	0.43	0.35
Locked environment needed	0.74 (0.13)	0.96 (0.14)	1.10	0.27	0.11
Police involvement appropriate	1.74 (0.23)	2.05 (0.22)	0.97	0.33	0.26
Attendance at job centre might be unhelpful	3.71 (0.17)	3.48 (0.17)	0.97	0.33	0.19
Bereavement counselling not indicated	2.55 (0.20)	2.46 (0.18)	0.32	0.75	0.82
Should be charged with criminal damage	0.33 (0.11)	0.43 (0.09)	0.69	0.49	0.02[2]
Should be remanded in custody	1.14 (0.23)	1.68 (0.27)	1.55	0.12	0.07
Difficult to establish rapport	3.10 (0.13)	2.85 (0.14)	1.33	0.19	0.26
Unlikely to comply	3.38 (0.14)	3.28 (0.12)	0.56	0.58	0.42
Family counselling not indicated	1.61 (0.18)	1.71 (0.18)	0.41	0.69	0.50
Do not refer to community self-help group	1.43 (0.18)	1.93 (0.21)	1.79	0.08	0.07
Offer genetic counselling	0.50 (0.12)	0.57 (0.12)	0.42	0.67	0.29
Antidepressant treatment not indicated	3.23 (0.18)	3.21 (0.20)	0.07	0.95	0.99

1. Scored 0–5, higher scores indicating agreement with statement about patient.
2. 'Significant' results.

adjusted for the possible confounding effects of sex of respondent using multiple-regression methods, and this did not alter materially the results presented below.

'Race'

The overall effect of the 'race' of the 'case' was found to be statistically significant when assessed irrespective of sex, by multivariate analysis of variance (MANOVA) with all 23 semantic differentials as the dependent variables ($F_{23,90}=1.71$; $P=0.04$). The full results for each semantic differential are shown in Table I. Tests of significance of the difference between scores were calculated by both parametric (*t*-test) and non-parametric methods (Wilcoxon's rank test) since some of the distributions of scores are skewed. Multivariate analysis of variance was used because the possibility of a type 1 error was increased by multiple hypothesis testing.

Sex

The overall effect of sex of the vignette, irrespective of race, was assessed using a MANOVA ($F_{23,90}=1.63$; $P=0.05$). Four of the items differed significantly at the 5% level (Table II). Although it was felt more appropriate to charge men with criminal damage, the responses indicated that this was not regarded as appropriate overall.

Interaction between sex and 'race'

As response to some items appeared to be influenced by the sex of the vignette *and* by race (Tables I, II), it might

TABLE II

Influence of sex of vignette on mean (s.e.m.) scores for semantic differentials

Statement about patient	*Male*	*Female*	t	P	*Wilcoxon*
Risk of violence to staff	2.83 (0.14)	2.40 (0.15)	2.14	0.03	0.05
Risk of violence to family	3.24 (0.12)	2.67 (0.14)	3.07	0.003	0.004
Neuroleptic treatment indicated	4.22 (0.12)	3.79 (0.18)	1.99	0.05	0.04
Should be charged with criminal damage	0.54 (0.13)	0.21 (0.06)	2.30	0.02	0.04

be reasonable, *a priori*, to suggest that black men might be subject to racial stereotypes more readily than black women. It is therefore necessary to examine the data for an interaction between sex and 'race'.

This hypothesis was examined using MANOVA. There was no statistical support for an interaction using all 23 items ($F_{23,88} = 1.04$; $P = 0.4$), although there was support for independent effects of race ($F = {}_{23,88} = 1.66$, $P = 0.05$) and sex ($F_{23,88} = 1.58$; $P = 0.07$). This result indicates that the effect of 'race' cannot be explained by the sex difference.

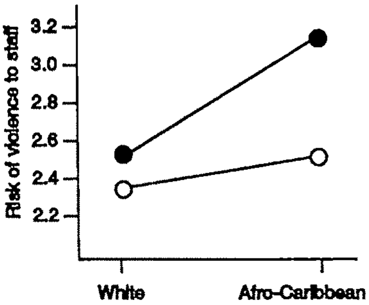


FIG. 1 The interaction between sex (●–● male, ○–○ female) and 'race' on increasing risk of violence to staff.

There was some indication, however, after thorough scrutiny of the data, that one item, 'Risk of violence to staff', illustrated an interaction (Fig. 1), with black males being regarded as potentially more violent. This could not be demonstrated statistically using an interaction term ($P = 0.25$), although this could have resulted from a lack of statistical power.

Diagnosis

Respondents were asked to give their three most likely diagnoses in order of preference. Schizophrenia was more commonly diagnosed when the hypothetical patient was white (Table III; $z = 1.54$; $P = 0.06$, one-tailed t-test) and there was a slight excess of acute reactive psychosis in the Afro-Caribbean group. When each diagnosis was considered in turn, the only group difference that reached significance was for cannabis psychosis (Wilcoxon's rank test, $P = 0.04$). Although cannabis psychosis was rarely chosen as a primary diagnosis in either group (Table III), it was more commonly given second or third place in the Afro-Caribbean group.

Table III also gives results for the item 'Risk of violence to staff' stratified by diagnosis. It can be seen that the Afro-Caribbean group still show higher scores within each diagnostic category. Although the small numbers prevent statistical analysis, the group difference illustrated here cannot be explained in terms of diagnostic preference.

There was no indication that the sex of the vignette influenced diagnosis.

Experience and 'race' of psychiatrist

The sample was divided into three groups according to the number of years' experience in psychiatry. We examined the following six items: risk of violence to staff and family, neuroleptic treatment indicated, duration of illness, and need for criminal proceedings including remand. A MANOVA showed that more experienced psychiatrists gave significantly and consistently lower scores ($F_{6,123} = 3.47$; $P = 0.003$). To examine whether the 'race' of the psychiatrists influenced their attitudes to the patient we asked them to state where they obtained their qualifications in medicine. It was felt that a straightforward question about ethnicity would prime the respondents to be more conscious of their attitudes towards different 'racial' groups and so this question was not asked. There was no statistical evidence to support any difference between the attitudes of foreign-trained and British-trained graduates.

Discussion

The results support the notion that the 'race' of a patient influences clinical predictions and attitudes of practising psychiatrists. Furthermore, it is possible to extrapolate a stereotype of a black person presenting with a psychosis. This stereotype consists of an illness of short duration, more likely to lead to violence, in which criminal proceedings are slightly more appropriate, and in which neuroleptic medication is less likely to be necessary. It most easily fits the rubric of brief/acute reactive psychosis and may be especially strong in a male patient. The

TABLE III

Frequency of most favoured diagnosis and perceived risk of violence, by race of case vignette

Diagnosis	*Frequency of diagnoses*		*Risk of violence to staff*[1]	
	Afro-Caribbean	*white*	*Afro-Caribbean*	*white*
Schizophrenia	20 (38.5%)	32 (49.2%)	2.75 (0.24)	2.66 (0.24)
Affective disorder	11 (21.2%)	12 (18.5%)	3.28 (0.24)	2.77 (0.33)
Cannabis psychosis	4 (7.7%)	5 (7.7%)	3.00 (0.41)	2.80 (0.80)
Acute reactive psychosis	16 (30.8%)	13 (20.0%)	2.87 (0.30)	2.15 (0.32)
Pathological grief reaction	1 (1.9%)	3 (4.6%)	-	-
Schizotypal personality	0	0		
Total	52	65		

1. Mean score (s.e.m.) on semantic differential.

diagnosis of cannabis psychosis was considered more often in the Afro-Caribbean group, although it was infrequently applied. More importantly there was a suggestion that schizophrenia was *less* likely to be diagnosed in the Afro-Caribbean group. This was unexpected considering claims that schizophrenia is overdiagnosed in blacks in the USA (Loring & Powell, 1988). The 'race' of the vignette did not appear to determine whether treatment should be community or hospital orientated, nor did it lead to unequal estimates of depression.

The study sample was intended to be representative of consultant psychiatrists working in Britain today and the response rate was 73%. The group were very experienced (mean 21 years) and this may limit the generalisability of the data; junior psychiatrists will also have an influence on diagnostic and management decisions. In addition, older psychiatrists appeared to predict less violence and a better outcome. A further limitation on the generalisability is the use of a single case description, as particular facets of the case could have elicited (or concealed) the psychiatrists' racial stereotype.

The main aim was to examine the differences between scores which can be attributed to the 'race' of the vignette. This was interpreted as evidence for a racial stereotype. The mean responses on neuroleptic treatment, for instance, showed that on average the psychiatrists thought that neuroleptics were indicated for both black and white vignettes. The same argument applies to the criminal damage item, as few psychiatrists thought legal proceedings were justified. We stress that it is the presence of a difference between the scores, irrespective of its size or absolute value, which supports the notion of a racial stereotype.

It should be emphasised that the current study examined attitudes to a hypothetical case and may not be a true reflection of actual practice. Respondents may also have been aware that attitudes to 'race' were under study and so racist views would be modified in the direction of social desirability. However, this would not explain the increased likelihood of violence and charging with criminal damage in the black group. Moreover, gender of the case vignette produced an effect in a predictable direction (i.e. women less violent, less in need of custodial care), lending indirect support for the validity of the method.

Among the aims of the study was to examine interactions between 'race' and other variables including sex of vignette. Unfortunately, the low statistical power of the small sample size will increase the chance of type II errors (false negative). For example, the effect of 'race' on perceived violence was approximately five times greater in the male compared with the female vignette (Fig. 1) but failed to reach conventional levels of statistical significance. We were even more restricted in examining interactions between black psychiatrists and black cases, as we chose not to ask subjects a question on ethnicity, and there were few psychiatrists who trained abroad and none who trained in the Caribbean. On this point, Loring & Powell (1988) found that both black and white psychiatrists in the US were biased similarly by the 'race' of a case vignette.

Racism and psychiatry

Commentators who have emphasised the importance of racist attitudes in psychiatry (Burke, 1984) have presumed a somewhat different racial stereotype to that described here. While sharing some features in the above, it also includes overdiagnosis of schizophrenia, increased use of custodial care, and heavy doses of neuroleptic drugs (Littlewood & Cross, 1980; Littlewood & Lipsedge, 1981). However, in this study there was no indication of a greater readiness to detain patients compulsorily or to manage them on a locked ward merely on the grounds of 'race'. Also, the results suggest that the increased prevalence of schizophrenic disorders recorded in a number of studies (McGovern & Cope, 1987; Harrison *et al*, 1988) cannot readily be ascribed to tendencies among British psychiatrists to overdiagnose schizophrenia in black people: a trend in the opposite direction was noted. The stereotype elicited here is consistent with the influential early descriptions in the literature of psychotic reactions in Afro-Caribbeans (Littlewood & Lipsedge, 1981).

At first glance, some aspects of the stereotype such as diagnostic preference may seem reassuring. However, we believe that any racial stereotype has harmful consequences, especially where it interferes with clinical judgement. For example, the duration of psychosis in Afro-Caribbeans is no shorter than in whites (Harrison *et al*, 1988), and failure to acknowledge the true prognosis may lead to inappropriate management. Finally, a tendency to ascribe psychotic reactions to cannabis may also deflect from proper management (Pilowsky & Moodley, 1989). The criteria for the diagnosis of 'cannabis psychosis' are poorly defined and at present, the best conducted study in this field pointed to a possible role of cannabis in increasing the risk of schizophrenia (Andréasson *et al*, 1987).

Conclusion

The racial stereotype elicited here contains some aspects that most people would regard as implying inferiority; for instance, the attribution of increased violence and the appropriateness of criminal proceedings in Afro-Caribbeans. However, the other aspects are not so clearly pejorative. The evidence for a racial stereotype provided here, irrespective of any implied inferiority, supports the view that race-thinking is prevalent among British psychiatrists. Race-thinking distorts all social interactions, including the psychiatric assessment, and thus undermines the 'objectivity' of psychiatric diagnosis and management. There is no indication that interpersonal aspects of care such as ability to establish a rapport, counselling and predicted compliance are prejudiced on grounds of race. Rather there is a tendency to expect brief, perhaps cannabis-influenced, violent psychotic reactions of limited duration and in less need of neuroleptic medication.

Although some commentators have argued that racism in psychiatrists merely reflects racism in British society, the content of the stereotype may be more influenced by professional literature and training. We hope that by highlighting the nature of this stereotype its unwitting use may be prevented.

The term 'racism' may be applied to some British psychiatrists, but such accusations seldom change beliefs or behaviour for the better. We suggest that race-thinking is a wider problem, and though less obvious and obnoxious than outright ideological racism, it is still an unseen and damaging influence.

Acknowledgements

This research was funded by the Department of Health through the General Practice Research Unit. We thank all the respondents for their co-operation. We would like to acknowledge the helpful criticism provided by Jonathan Gabe, Louis Appleby, and Tom Fahy. Dr David is supported by the MRC.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Andréasson, S., Allebeck, P. Engstrom, A., *et al* (1987) Cannabis and schizophrenia. a longitudinal study of Swedish conscripts. *Lancet*, *ii*, 1483–1486.

Burke, A. (1984) Racism and psychological disturbance among West Indians in Britain. *International Journal of Social Psychiatry*, **30**, 50–68.

Cochrane, R. & Bal, S. S. (1989) Mental hospital admission rates of immigrants to England: a comparison of 1971 and 1981. *Social Psychiatry and Psychiatric Epidemiology*, **24**, 2–11.

Cox, J. L. (ed) (1986) *Transcultural Psychiatry*. London: Croom Helm.

Dean, G., Walsh, D., Downing, H., *et al* (1981) First admissions of native-born immigrants to psychiatric hospitals in South-East England 1976. *British Journal of Psychiatry*, **139**, 506–512.

Department Of Health And Social Security (1987) Medical and dental staffing prospects in the NHS in England and Wales in 1986. *Health Trends*, **19**, 1–7.

Fahy, T. A. (1989) The police as a referral agency for psychiatric emergencies. *Medicine, Science and the Law*, **29**, 315–322.

——, Bermingham, D. & Dunne, J. (1987) Police admissions to psychiatric hospitals: a challenge to community psychiatry. *Medicine, Science and the Law*, **27**, 263–268.

Fernando, S. (1986) Depression in ethnic minorities. In *Transcultural Psychiatry* (ed. J. L. Cox). London: Croom Helm.

Ford, M. & Widiger, T. A. (1989) Sex bias in the diagnosis of histrionic and antisocial personality disorders. *Journal of Consulting and Clinical Psychology*, **2**, 301–305.

Glover, G. R. (1989) The pattern of psychiatric admissions of Caribbean-born immigrants in London. *Social Psychiatry and Psychiatric Epidemiology*, **24**, 49–56.

Harré, R. & Lamb, R. (1983) *The Encyclopedic Dictionary of Psychology*. Oxford: Blackwell.

Harrison, G., Owens, D., Holton, A., *et al* (1988) A prospective study of severe mental disorder in Afro-Caribbean patients. *Psychological Medicine*, **18**, 643–657.

Husband, C. (1982) Introduction: 'race', the continuity of a concept. In *'Race' in Britain: Continuity and Change* (ed. C. Husband), pp. 11–23. London: Hutchinson

Ineichen, B., Harrison, G. & Morgan, H. G. (1984) Psychiatric hospital admissions in Bristol I: geographical and ethnic factors. *British Journal of Psychiatry*, **145**, 600–604.

Lewis, G. & Appleby, L. (1988) Personality disorder: the patients psychiatrists dislike. *British Journal of Psychiatry*, **153**, 44–49

Littlewood, R. (1988) Community-initiated research: a study of psychiatrists' conceptualisations of 'cannabis psychosis'. *Psychiatric Bulletin*, **12**, 486–488.

—— & Cross, S. (1980) Ethnic minorities and psychiatric services *Sociology Health and Illness*, **2**, 195–201.

—— & Lipsedge, M. (1981) Some social and phenomenological characteristics of psychotic immigrants. *Psychological Medicine*, 11, 289–302.

—— & —— (1988) Psychiatric illness among British Afro-Caribbeans. *British Medical Journal*, **296**, 950–951.

Loring, M. & Powell, B. (1988) Gender, race and DSM-III: a study of the objectivity of psychiatric diagnostic behaviour. *Journal of Health and Social Behaviour*, **29**, 1–22.

McGovern, D. & Cope, R. (1987) The compulsory detention of males of different ethnic groups, with special reference to offender patients. *British Journal of Psychiatry*, **150**, 505–512.

Mercer, K. (1984) Black communities' experience of psychiatric services. *International Journal of Social Psychiatry*, **30**, 22–27.

Onyango, R. S. (1986) Cannabis psychosis in young psychiatric inpatients. *British Journal of Addiction*, **81**, 419–423.

Pilowsky, L. & Moodley, P. (1989) 'Cannabis psychosis'. *Psychiatric Bulletin*, **13**, 148.

Rwegellera, G. G. (1977) Psychiatric morbidity among West Africans and West Indians living in London. *Psychological Medicine*, **7**, 317–329.

*Glyn Lewis, MSc, MRCPsych, *Lecturer in Psychiatry, General Practice Research Unit, Institute of Psychiatry*; Caroline Croft-Jeffreys, BA, *Research Assistant, General Practice Research Unit*; Anthony David, MPhil, MRCP, MRCPsych, *Lecturer in Psychiatry, Institute of Psychiatry, De Crespigny Park, London SE5 8AF*

*Correspondence

British Journal of Psychiatry (1990), 157, 416–420

A Comparative Study of Psychiatric Services in Japan and England

YOSHIO MINO, RYOSEI KODERA and PAUL BEBBINGTON

Psychiatric services in Japan and England are compared using government statistics. In Japan, the number of in-patients per 100 000 population has increased from the 1950s, while that of England has decreased since 1954. Since 1972 the prevalence of in-patients has been higher in Japan than in England. The admission rate is lower in Japan than in England, and there are more long-stay patients. Most Japanese in-patients are admitted compulsorily, whereas most are admitted voluntarily in England. The attendance at out-patient clinics is higher in Japan than in England, but there are far fewer day-hospital places in Japan. Differing government policies are the main reason for these differences.

Many problems exist in the psychiatric services in Japan. Criticisms have recently been made by a British psychiatrist (Fahy, 1987) and a journalist (Cohen, 1988). In addition, the International Commission of Jurists sent two expert Missions, one in 1985 (also supported by the International Commission of Health Professionals) and the other in 1988, which reported in detail on problems in the Japanese situation (Harding *et al*, 1986; International Commission of Jurists' Mission to Japan, 1988). In Japan itself, psychiatric services have been criticised from the viewpoints of psychiatry (Nishizono, 1987; Ogawa *et al*, 1987), public health (Aoyama, 1983) and the law (Totsuka *et al*, 1986). These criticisms can be condensed into three: (1) the harmful effects of large, closed mental hospitals; (2) the poor community care services; and (3) the infringement of the human rights of mentally ill patients in mental hospitals.

In response to these criticisms, the Japanese Ministry of Health and Welfare made recommendations (Koseisho Seishin Hoken Ka, 1988) which led to the reform of the Mental Health Act in 1987 (becoming law in 1988). According to the new law, a rehabilitation system for mentally-ill patients was to be developed, and such patients' human rights were respected. However, there have been difficulties implementing the law since few studies have identified the problems in the psychiatric services clearly.

The situation in Japan can be contrasted with that of other industrial countries such as England. After the Second World War psychiatric services changed notably in England from being hospital-centred to being more community-based. The advent of the NHS and the introduction of psychotropic drugs made it easier to care for patients in the community (Wing, 1985; Gelder *et al*, 1986). At its best, England's mental health system is highly regarded all over the world (Salokangas *et al*, 1985). It is valuable therefore to compare the psychiatric services in Japan with those in England in order to cast light on the former.

Method

The study is based on available government statistics from Japan and England. In Japan these were mainly obtained from the Ministry of Health and Welfare's Kanja Chosa (Patients' Survey) conducted on an annual census day (Koseisho Tokei Joho Kyoku, 1960–85), and are therefore based on patients in a randomly chosen 10% of all hospitals and 1% of all out-patient clinics (the total number of patients being estimated from these). The English statistics were obtained from the Mental Health Inquiry for England (Department of Health and Social Security, 1960–1984). Before 1970, statistics for England and Wales were combined, but since then, figures for England have been published separately. Thus, although our pre-1970 data are for both England and Wales, for convenience, we refer to them all as 'English', as in another comparative study (Salokangas *et al*, 1985).

We compare the following aspects of service use: rates of in-patients per 100 000 population, distributed by sex and age; rates of admission to mental hospitals per 100 000 population; the ratio of admissions to numbers of beds occupied (i.e. in-patients) on the annual census day (Wing, 1972); the duration of stay of in-patients (here we used data from another government survey (Koseisho, 1983) since adequate official government statistics were not available); the duration of stay of discharged patients (the available data for Japan were only for discharge from hospitals with over 19 beds); and rates of psychiatric out-patient and day-hospital attendance per 100 000 population.

In order to calculate the number of patients attending out-patient clinics in Japan, the number of attendances on the annual census day was multiplied by 250, the usual number of working days a year for most Japanese people. The exact number was unobtainable from government statistics. We were also unable to get data on attendance at day-hospitals in Japan.

Results

A comparison of the numbers of in-patients on annual census days is shown in Fig. 1. In Japan, the in-patient rates increased from 32 per 100 000 total population in 1953 to 260 in 1984. In England, by contrast, they decreased from

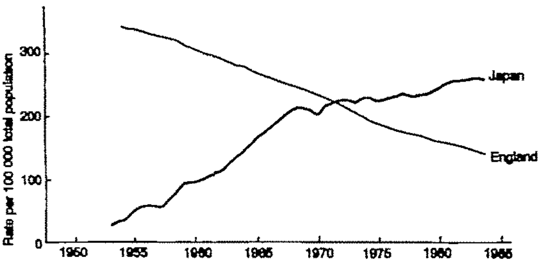


FIG. 1 Rates of in-patients per 100 000 population in Japanese and English mental hospitals on annual census days.

344 in 1954 to 141 in 1984. Since 1972, the rate in Japan has been higher than that in England and the difference between the countries has continued to increase since then.

A comparison of in-patients by age and sex is shown in Table I. The rates for Japan increased in both sex groups, while those for England decreased. In Japan, there are more men than women in mental hospitals, while in England the opposite is true, a tendency similar to that between Finland and England (Salokangas *et al*, 1985). This can be interpreted in two ways. Firstly, since women usually play a major role in the home in Japan, it may be easier for women with mental illness to maintain this in the absence of adequate social support systems than men, whose roles, largely located outside the home, may be more demanding. Secondly, there are, and have been, higher rates of elderly in-patients in England and, since there are more women than men in the elderly (over 65 years) population in England, this may also account for the difference.

Regarding age-specific rates, rates of bed-occupancy (i.e. in-patients) decreased consistently in all age-groups in England. Although rates decreased among those under 35 years in Japan, they increased in groups over 45 years.

TABLE I
Total, sex and age-specific rates per 100 000 population for in-patients in Japan and England

	Japan				*England*			
	1970	*1975*	*1980*	*1984*	*1970*	*1975*	*1980*	*1984*
Total	200	220	245	258	233	188	161	141
Sex:								
male	239	264	288	298	206	166	–	–
female	163	181	203	220	260	209	–	–
Age: years								
15–24	142	133	96	81	48	50	36	29
25–34	324	310	297	264	110	88	74	65
35–44	368	413	412	396	190	131	102	79
45–54	302	362	425	471	292	227	156	114
55–64	244	288	356	406	391	311	253	205
65–74	186	232	329	356	599	479	388	352
75+	141	212	406	484	1208	918	870	775

TABLE II
Ratio of Japanese : English in-patients in given years by sex and age

	1970	*1975*	*1980*	*1984*
Total	0.86	1.17	1.52	1.83
Sex:				
male	1.16	1.59	–	–
female	0.63	0.87	–	–
Age: years				
15–24	2.96	2.66	2.67	2.79
25–34	2.95	3.52	4.01	4.06
35–44	1.94	3.15	4.04	5.01
45–54	1.03	1.59	2.72	4.13
55–64	0.62	0.93	1.41	1.98
65–74	0.31	0.48	0.85	1.01
75+	0.12	0.23	0.47	0.62

Table II shows the ratio of Japanese to English in-patients. The ratios increased with time in all ages except the group under 24 years, and, by 1984, the ratios exceeded four in the groups from 25 to 54 years.

A comparison of admission rates to mental hospitals per 100 000 total population is shown in Fig. 2. In general, the rates in Japan have been around half of those in England. Before 1980, trends in both countries were similar, the rates increasing gradually in the 1960s to a plateau in the 1970s. After 1980, they remained stable in Japan, but increased in England.

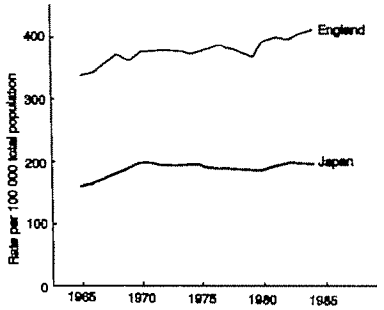


FIG. 2 Rates of admissions to mental hospitals per 100 000 population in Japan and England.

The ratios of admissions to numbers of beds occupied are shown in Table III. This ratio has increased steadily in England but has gradually decreased in Japan since 1970. In 1984, the ratio in England was four times that in Japan.

With regard to length of stay of in-patients in Japan, because of the limited information, we can only give (1) the rates of discharged patients per 100 000 population broken down by the duration of stay in hospital (Fig. 3), and (2) the distribution of length of stay of in-patients, which is available in Japan only for 1983 (Table IV). Figure 3 clearly shows that patients are discharged considerably earlier in England than in Japan. In England, the rate of patients discharged within one month of admission has

TABLE III

The ratio of admissions : numbers of beds occupied in given years in Japan and England

Year	*Japan*	*England*
1965	0.95	1.28
1970	0.98	1.61
1975	0.86	2.01
1980	0.75	2.41
1984	0.74	2.90

increased steadily, whereas in Japan the increase is recent. Table IV shows how in Japan in 1983, almost 50% of in-patients had been in hospital more than five years and only 25% had been admitted less than one year previously. In England in 1984, the proportion of in-patients resident over five years had decreased by 36%, while those resident under one year had increased by 40%.

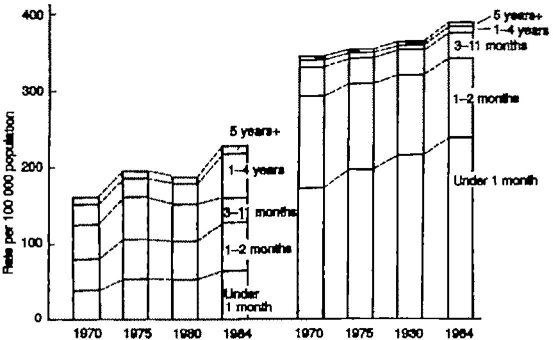


FIG. 3 Discharge rates per 100 000 population from mental hospitals in Japan and England broken down by length of stay in hospital.

These results suggest that in England the numbers of 'old' long-stay patients (over five years) have decreased and the length of stay of discharged patients has considerably reduced. However, in Japan the 1983 data suggest that a large percentage of patients remain in hospital for more than five years. This must mean that many Japanese mental patients are still at risk of 'institutionalism' (Wing & Brown, 1970).

TABLE IV

Length of stay in hospital of in-patients in Japan and England

	Japan	*England*			
	1983	*1970*	*1975*	*1980*	*1984*
Length of stay in hospital: %					
<1 year	24.0	28.2	31.2	36.0	39.9
1–2 years	16.5	13.6	13.7	15.2	15.3
3–4 years	10.1	58.1	7.1	8.2	8.7
5+ years	47.8		47.9	40.7	36.0
unknown	1.6	–	–	–	–
total	100.0	100.0	100.0	100.0	100.0

The legal category of admission to mental hospital is also relevant. Before 1988 in Japan, there were three types of formal (compulsory) admission: compulsory admission by the governor under article 29 of the Mental Health Act; compulsory admission with the consent of the person responsible for care under article 33; and provisional compulsory admission under article 34. Most admissions have been formal and under articles 29 and 33. For example, the proportion of compulsory admissions in 1978 was estimated to be over 80% (Totsuka *et al*, 1986), and, according to a later survey (Koseisho, 1983), was 93.6% (80.1% of in-patients admitted under article 33 and 13.5% under article 29). In contrast, the proportion of informal (voluntary) admissions in England was 82.9% in 1978, increasing to 92.9% in 1984. Moreover, there was no form of appeals procedure for patients detained under article 29 or 33. This raised questions both of the patients' human rights and of policies of treatment. In response, the government reformed the Mental Health Act in 1988, recommending the general adoption of voluntary admission, and regulated the form of appeals procedure for patients detained. In addition, a Local Psychiatric Review Board was established to consider whether an in-patient should remain in hospital and whether he or she has received adequate treatment (Koseisho Seishin Hoken Ka, 1988; International Commission of Jurists' mission to Japan, 1988).

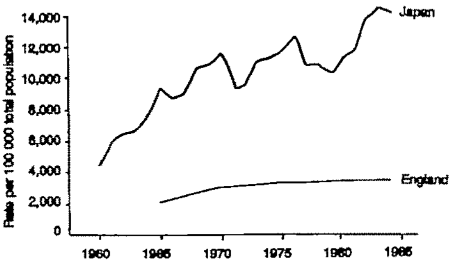


FIG. 4 Rates of psychiatric out-patient attendances per 100 000 population in Japan and England.

A comparison of psychiatric out-patient attendances in Japan and England is shown in Fig. 4. Since the 1960s, the attendance rate in Japan has been more than that in England. However, we must consider two points when interpreting this: (1) the Japanese data are a relatively crude estimate – the number of attendances on the annual census day being simply multiplied by 250; and (2) in England, out-patient visits do not give a correct picture of non-residential care, since there are more attendances at day-hospitals than at out-patient clinics and a large number of patients are also treated by general practitioners.

The number of attendances at day-hospitals increased throughout the 1970s in England, the rate of attendance per 100 000 population being 7 282 in 1982. Non-hospital day-centres have also played an important role in community care. However, these are not included in Fig. 4, as in Japan there are no adequate equivalent data. Indeed, there were only 125 officially recognised day-care facilities in the whole country in 1986 (Koseisho Seishin Hoken Ka, 1988).

Discussion

This study is based on government statistics from the two countries which, in general, enable comparisons to be made. However, the data for Japan are less comprehensive than those for England, for example, with regard to the duration of in-patient stay and the attendance at psychiatric out-patient clinics and day-hospitals. However, these differences in the quality of the statistics can not explain the differences in the psychiatric services.

There are three factors that may explain the differences in psychiatric services in Japan and England: (1) the differences in psychiatric morbidity; (2) the differences in government policy; and (3) the differences in public attitudes towards mental illness (Salokangas *et al*, 1985).

On the issue of differences in psychiatric morbidity, although more epidemiological studies are required, the differences in psychiatric services between Japan and England can hardly be explained by this factor. Kato (1969) for example reviewed epidemiological surveys by the Japanese Ministry of Health and Welfare and found no increase in the prevalence rates of mental disorder between 1954 and 1963, in spite of the rapid increase of in-patient rates during this period.

Differences in government policy have clearly contributed to the differences in services. In Japan, the history of mental-health services can be divided into three stages, relating to the enactment of, and changes in, the Mental Health Act, pre-1950, 1950–65 and post-1965. Before 1950, most mentally ill patients were cared for in the community, often not being treated by psychiatric specialists and sometimes virtually detained in the home by family members. In 1950, the Mental Health Act was passed and the basis of the Japanese psychiatric service was established. The incarceration of mentally ill patients by family members was prohibited, and formal (involuntary) admission was legally established and regulated. The government regarded an increase of residential facilities for mentally ill patients as a priority and supported private mental hospitals financially. In consequence, the number of beds for mentally ill patients increased, mainly in private mental hospitals, and the number of resident in-patients began to grow.

After the revision of the Mental Health Act in 1965, the concept of community mental health care was introduced. Under the reformed act, public health centres were designated as the primary basis of mental health services, and mental health centres were established in each prefecture. However, these facilities have not shown good results, mainly because of poor implementation, an insufficient financial base and a consequent shortage of staff. Consequently, psychiatric services in Japan have been mainly hospital-centred, and the numbers of resident in-patients have increased throughout this period (Koseisho Seishin Hoken Ka, 1988; Okagami & Wada, 1985). A fourth stage has just begun following a further reform of the Mental Health Act in 1988.

The history of psychiatric services in England has been described in detail elsewhere (Wing, 1985; Gelder *et al*, 1986). Despite serious criticisms (House of Commons, 1985; Audit Commission Report, 1986), community care has been at least relatively well established in England, and this constitutes the major difference between the two countries.

In the UK, the National Health Service (NHS) has ensured that virtually all mental hospitals and day-hospitals are public. On the other hand, in Japan, 85% of mental hospitals are private and this is a disadvantage for changing policy from hospital-centred to community-based. Moreover, there has been little central government control over the private mental hospital system. However, the Japanese government recently began to play a leading part in the mental health system and to control private mental hospitals. The amendment of the Mental Health Act in 1988 was an expression of this trend.

The results of this study imply that attitudes towards mental illness in Japan are less tolerant than those in England. There are more compulsory admissions and more closed wards in Japan and this situation has been accepted both by psychiatrists and by the general public. Through the new Mental Health Act the Japanese government intends to help reduce the stigma of mental illness, and legal constraints on compulsory admission should become stricter. This is both a cause and a reflection of changing attitudes among Japanese people.

Conclusions

Of the three possible explanations of the differences between English and Japanese psychiatric services, the differences in government policy seem to be the most important, and this is closely related to the attitudes of people towards mental illness.

The data on which this study is based can not describe the quality of care and of life for the mentally ill. An analysis of such factors is essential in developing community-based care for mentally ill patients. In 1988, the amendment of the Mental Health Act in Japan gave government backing to psychiatrists and other mental health professionals to develop better community care for patients. However, among Japanese psychiatrists and other professionals, there is a fear that deinstitutionalisation may occur without the establishment of a community health-care system, leading to homelessness and vagrancy among the mentally ill. None the less, we believe that a community social support system for mentally ill patients can be developed. Such a project can only benefit from comparisons with those of other countries. In particular, Japan can learn both from the successes and failures of England's attempt over the last 30 years to build a better, more community-based system.

References

Aoyama, H. (1983) Public health and medical practice and psychiatric administration (in Japanese). *Seishin-iryo*, 11, 4–14.

Audit Commission (1986) *Making a Reality of Community Care*. London: Her Majesty's Stationery Office.

Cohen, D. (1988) *Forgotten Millions*. London: Paladin Grafton Books.

Department Of Health And Social Security. *In-patient Statistics from the Mental Health Enquiry for England (and Wales)* (1960–1984). London: Her Majesty's Stationery Office.

Fahy, T. (1987) Japan, liberty & lunacy. *The Bethlem and Maudsley Gazette*, 34, 13–15.

Gelder, M., Gath, D. & Mayou, R. (1986) Psychiatric services. In *Oxford Textbook of Psychiatry* (eds M. Gelder, D. Gath & R. Mayou). Oxford: Oxford University Press.

Harding, T. W., Schneider, J. J., Visotsky, H. M., *et al* (1986) *Human Rights and Mental Patients in Japan*. Geneva: International Commission of Jurists.

House Of Commons (1985) *Second Report from the Social Services Committee, Session 1984–1985: Community Care*. London: Her Majesty's Stationery Office.

International Commission Of Jurists' Mission To Japan (1988) *Preliminary Report and Recommendations*. Geneva: ICJ.

Kato, M. (1969) Psychiatric epidemiology surveys in Japan: The problem of case finding. In *Mental Health Research in Asia and the Pacific* (eds W. Caudill & T. Lin). University of Hawaii: East–West Center Press.

Koseisho (1983) *Seishin Eisei Jittai Chosa Hokoku no Gaiyo* (in Japanese). Tokyo: Koseisho.

Koseisho Seishin Hoken Ka (1988) *Wagakuni no Seishin Hoken* (in Japanese). Tokyo: Kosei Kanko Mondai Kenkyukai.

Koseisho Tokei Joho Kyoku (1960–1984) *Kanja Chosa (Patient Survey)* (in Japanese). Tokyo: Koseisho.

Nishizono, M. (1984) Overview of mental hospitals in Japan (in Japanese). *Nisseikyo-Zasshi*, 3, 47–55.

Ogawa, K., Miya, M., Watari, A., *et al* (1987) A long-term follow-up study of schizophrenia in Japan – with special reference to the course of social adjustment. *British Journal of Psychiatry*, 151, 758–765.

Okagami, K & Wada, S. (1985) Community mental health services in Japan: the role of public health centers, mental health centers, and the National Institute of Mental Health. *Journal of Mental Health*, 32, 141–146.

Salokangas, R. K. R., Der, G. & Wing, J. K. (1985) Community psychiatric services in England and Finland. *Social Psychiatry*, 20, 23–29.

Totsuka, E., Mitsuishi, T. & Kitamura, Y. (1986) Mental health and human rights: illegal detention in Japan. In *Psychiatry Law and Ethics* (eds A. Carmi, S. Schneider & A. Hefez). Berlin: Springer-Verlag.

Wing, J. K. (1972) Principle of evaluation. In *Evaluating a Community Psychiatric Service: The Camberwell Register, 1964–1971* (eds J. K. Wing & A. M. Hailey). Oxford: Oxford University Press.

—— (1985) Mental health services in United Kingdom. In *Psychiatry, The State of the Art 7, Epidemology and Community Psychiatry* (eds P. Pichot, P. Berner, R. R. Wolf & K. Thau). New York: Plenum Press.

—— & Brown, G. W. (1970) *Institutionalism and Schizophrenia, A Comparative Study of Three Mental Hospitals 1960–1968*. London: Cambridge University Press.

Yoshio Mino, MD, *Instructor, Department of Public Health, Kochi Medical School, Japan*; Ryosei Kodera, MD, *Chief, Kochi Prefectural Motoyama Health Centre, Japan*; *Paul Bebbington, MA, MB, PhD, MRCP, MRCPsych, *Reader in Social and Epidemiological Psychiatry, MRC Social and Community Psychiatry Unit, Institute of Psychiatry, London*

*Correspondence

British Journal of Psychiatry (1990), 157, 421–424

Mass Sociogenic Illness by Proxy: Parentally Reported Epidemic in an Elementary School

SIMON WESSELY and CHRISTOPHER J. WARDLE

"In a cluster of illness reported among students at an elementary school parents mentioned many signs and symptoms including headache, pallor, dark circles under the eyes, nausea, and vomiting – which they attributed to exposure to recurrent leaks of natural gas at the school. It is likely that the parents spread among themselves the notion of toxic exposure at the school. A questionnaire revealed no spatial clustering, but increased reports of symptoms were related to intense media coverage. A thorough environmental and epidemiological investigation was negative, there being no evidence of a continuing gas leak or other potential causes. At a strictly biological level, the complaints in this reported 'cluster' apparently represented the sporadic occurrence of common childhood illnesses. The possibility of an epidemic from toxic exposure at the school caused intense parental concern and led to a major public health problem. The established term 'mass sociogenic illness' seems inapplicable here because complaints did not come principally from the students and the apparent epidemic illness was not transmitted among them. The term 'mass sociogenic illness by proxy' is proposed to describe this incident, in which transmission in one group (the parents) resulted in reports of an epidemic in another group (students)."

The summary quoted above is from an article by Philen *et al* (1989). The present authors were invited to comment upon the study.

Simon Wessely

This paper demonstrates that parental concerns are a potent cause of outbreaks of 'illnesses', and especially the group episodes often called 'mass hysteria'. I find the paper convincing, and am pleased to see it in a prestigious journal such as the *Lancet*. An academic purist could be less welcoming, pointing out that the authors are not reporting anything particularly new or surprising. It is certainly true that Mausner & Gezon (1967) described a similar epidemic of parental concern about a non-existent epidemic, in that case non-gonorrhoea. In fact, the purist might suggest that the current report is not an episode of mass hysteria at all, since no actual epidemic, defined by time/space clustering, occurred, nor did the participants regard themselves as ill or seek medical attention – all of which are necessary for an episode to be considered as 'mass hysteria' (Wessely, 1987). Instead, it should be classified as a further example of a false group rumour, as in such classics of the literature as the "Phantom Slasher of Taipei", the "Great Seattle Windscreen Pitting Epidemic", and the "Phantom Anaesthetist of Mattoon" (see Wessely, 1987). Nevertheless, these are minor objections to a well constructed and timely report.

What else does the paper tell us? In 1987, I reviewed 52 episodes of mass hysteria, and noted the change that has occurred in their content over the years and centuries. Participants no longer talked about demons and possession states, but now about viruses, mystery gases, and toxins. Since 1987, a further eight reports have been published, and this is the ninth. Environmental toxicity remains the principal explanation, and although some continue to be almost random events (Elkins *et al*, 1988; Ruiz & Lopez, 1988), most follow 'exposure' to toxic chemicals, either real or imaginary, (Goh, 1987; Sinks *et al*, 1989; Selden, 1989; O'Donnell *et al*, 1989; Simon *et al*, 1990) or environmental pollution (Araki & Honma, 1986). We are no longer oppressed by devils, but by allergies, pollution, and viruses.

In this paper, the authors obviously do not share the group belief that the so-called epidemic was due to toxic gas. Instead, they attribute the outbreak to 'stressors' within the institution – in this case, the failure of new classrooms to arrive on time. They suggest that all such outbreaks must reflect stress or psychopathology within the groups involved, and indeed are not alone in that assumption (see Sirois, 1974, for instance). However, this is not always justified. The failure of new classrooms to arrive is surely one of the many tribulations that beset every school in Britain and the USA every week. Instead, many (but certainly not all) episodes of mass short-term anxiety often have very trivial causes – such events can begin in an unpredictable, idiosyncratic fashion and, unlike the more serious chronic episodes, do not always indicate any particular psychopathology or social stress. The causes of such phenomena lie less within the institution, and even more rarely within the individual, but are to be found in the wider social context. Fashions in illness change, but will always reflect the preoccupations of the age. The preoccupations behind this and other outbreaks are not worries about facilities in our schools, deplorable though they may be, but a wider, shared belief in the deteriorating quality of the environment in which we live. This may, of course, be an appropriate concern, but how often are such factors a direct cause of ill-health? Very rarely – instead, it is more likely these illnesses result not from the actual change in the environment, but from the concern it engenders. The paradox of health is that as we are objectively becoming healthier and living longer, our perception of our health becomes worse, and the number of symptoms of which we suffer increases (Barsky, 1988). Discussing the related 'sick building syndrome' Bardana (1986) identified the major cause as the growing fear of our surroundings and environment – "to the extent that the sick building syndrome is an expression of our anxieties, it is a social problem".

Even if all this is true, does it matter? Does it make any difference that the parents probably misattributed the cause of their children's distress? After all, the 'epidemic' was short-lived, and no one suffered untoward consequences. Just as diabolical possession could be relatively benign, so long as the demon could be successfully exorcised, so are most of the modern episodes, and the authors suggest that one of the characteristics of mass sociogenic illnesses is the 'benign morbidity'. This is so usually, but not always. Returning to demonology, remember the Devils of Loudun and the Witches of Salem – if social validation is granted to those affected, and is continued, then the transient disturbances may become serious and severe. All seemed to have ended well at the high school in question, but what if the school authorities contacted not the sensible researchers represented here, but a less critical specialist in a field such as 'clinical ecology' (American College of Physicians, 1989). The same symptoms that the authors ascribe to the general level of non-specific illness in the community could then have become proof of 'environmental hazard', and susceptible children (which really means susceptible parents) would be on the way to a lifetime of ill-health. The authors cite the detrimental effect of media coverage – imagine the effect of media coverage of this episode if the doctors had accepted the parental explanation. In a polemical essay, Bell (1989) has pointed out the chaos and unnecessary ill-health that resulted from the well meaning collusion of doctors, journalists, sufferers and courts during the epidemic of 'repetitive strain injury' that recently swept across Australia.

Thus, one could state that this paper is merely rediscovering a forgotten wheel – one whose colouring has altered, but which still remains a wheel. Even the therapeutic approach they suggest should be adopted for future episodes, which is certainly appropriate and is as old as the condition itself – in 1787, Dr St Clare cured persistent collapse among Lancashire cotton workers by declaring the disease to be "merely nervous, easily cured and not introduced by cotton" (Hunter & Macalpine, 1964). Nevertheless, rediscovering the wheel, when it is in danger of being forgotten, is a worthwhile enterprise, for which the authors (and the *Lancet* editors) should be warmly congratulated. Perhaps the leading journals should take a solemn undertaking to publish similar reports every ten years, to protect us against our own gullibility.

Christopher J. Wardle

This paper raises the interesting issue of over 300 parents who made their children the subjects of a health *cause célèbre*, although they were not ill enough to be absent from school, noticed by their teachers, or taken to a medical practitioner. In fact, considerable influence of parental attitudes and behaviour on the development and progress of individual abnormal illness behaviour in children is observable in daily clinical practice. Only two other studies of mass abnormal illness behaviour in schools, out of 20 I was able to trace, have investigated family influences (Mohr & Bond, 1982; Small & Nicholl, 1982). Mohr found that a core of eight girls were responsible for triggering recurrent outbreaks of hysterical black-outs over two years in a Salford comprehensive school. These girls all had other behavioural and family problems; for instance, four lived with only one natural parent. Family preoccupation with illness, real or imagined, was prominent in this group, as well as in eight other girls who associated with them but had fewer and less severe attacks. The 44 girls and three boys who only had one or two attacks were contrasted in having stable, healthy families. Small (1982) found that the 34 children who were admitted in an acute episode of mass hysteria were much more likely to have experienced a death in their family (74%) or parental divorce (47%) than the 38 with symptoms but not admitted (40% and 11%) and the 110 asymptomatic children (38% and 10%).

Unfortunately, Philen *et al* seem to have missed an opportunity to follow-up these studies; they interviewed only five mothers who complained about their children, and the scanty information they give provides no explanation of these parents' active involvement nor justifies the use, in their title, of the term "by proxy". No children were interviewed.

Small & Nicholl (1982) noted that the arrival of reporters, police, and firemen with sirens blaring enhanced the spread of symptoms, as did panic measures by staff; it is curious and unfortunate that they called these services, rather than a doctor. Reports of new symptoms in the present study increased after mass media coverage and when parents picketed the school; at no stage was medical advice sought. The problem appears to have been prolonged, as in the Salford school, because of multiple, inconsequential, uncoordinated investigations; new, harmless little faults in the heating and ventilation systems kept being reported and corrected. Troubled illness behaviour is increased rather than relieved by repeated negative investigations, because these are interpreted by parents and child as meaning either that the doctors do not know what is wrong, or that they are hiding something. Either way, the implication is ominous.

How extreme the effects of media attention on a sensitised population can be, was reported by Moden *et al* (1983) from the West Bank of the Jordan. There, an epidemic of abdominal pain and other symptoms attributed to airborne toxins was started by the nauseating effect on a 17-year-old schoolgirl of a smelly lavatory. Sixty-two pupils in her school developed the same symptoms of pain, nausea, etc. within three hours. In the next three days, the symptoms spread to 10 other schools, eventually affecting 727 schoolgirls and 222 people in the surroundings, while 152 affected males included three Israeli soldiers.

Philen *et al* found an unusually high proportion of boys (47%) who reported symptoms, but most studies report a very high preponderance of adolescent females.

Throughout history, there are records of children being caught up in the madness of crowds (Mackay, 1841). Examples in the middle ages include the children's crusades, and the dancing mania; more recent cases are the dissociative and conversion phenomena associated with fundamentalist and revivalist sects. Perhaps the most extreme instances are to be found in the accounts of the 'witch mania' at its height in 17th-century Europe and America. Mackay reported that "the lying whim of a few sick children, encouraged by foolish parents, and drawn out by superstitious neighbours was sufficient to set a country aflame", referring to the horrifying epidemics in Blockula (Sweden), Huntingdon, and New England, where children developed gross hysterical symptoms in the belief they were bewitched. Their evidence led to many innocent people, including other children, being burnt at the stake, themselves convinced that they were possessed by the devil or had magic powers.

Reading *Extraordinary Popular Delusions and the Madness of Crowds* is a fascinating and salutary reminder of how easily any of us can be caught up and join the latest exciting craze and carry our children and friends with us. Education and science do not protect us; they may raise new fears of unknown environmental hazards, predisposing us to develop symptoms or new 'witch hunts' and 'manias'.

I hope this study will stimulate further investigation of the contribution of the attitudes and behaviour of both parents and other adults to the development of mass episodes of abnormal illness behaviour in children. Future studies need to clarify current misapprehensions about mass sociogenic abnormal illness behaviour: the age and sex distribution, the symptoms and the mode of transmission are much less stereotyped than has been suggested in these and other authors' attempts at classification.

References

American College of Physicians (1989) Position paper: clinical ecology. *Annals of Internal Medicine*, **111**, 168–178.

Araki, S. & Honma, T. (1986) Mass psychogenic systemic illness in school children in relation to the Tokoyo photochemical smog. *Archives of Environmental Health*, **41**, 159–162.

Bardana, E. (1986) Office epidemics. *The Sciences*, **26**, 39–44

Barsky, A. (1988) *Worried Sick: Our Troubled Quest for Wellness*. Boston: Little, Brown & Co.

Bell, D. (1989) "Repetition strain injury" – an iatrogenic epidemic of simulated injury. *Medical Journal of Australia*, **151**, 280–284.

Elkins, G., Gamino, L. & Rynearson, R. (1988) Mass psychogenic illness, trance states, and suggestion. *American Journal of Clinical Hypnosis*, **30**, 267–275.

Goh, K. (1987) Epidemiological enquiries into a school outbreak of an unusual illness. *International Journal of Epidemiology*, **16**, 265–270.

Hunter, R. & MacAlpine, I. (eds) (1964) An epidemic of hysterics. In *Three Hundred Years of Psychiatry*, p. 506 Oxford: Oxford University Press.

Mackay, C. (1841) *Extraordinary Popular Delusions and the Madness of Crowds*. (Reprinted 1932) New York: L. C. Page & Co.

Mausner, J. & Gezon, H. (1967) Report on a phantom epidemic of gonorrhoea. *American Journal of Epidemiology*, **85**, 320–331.

Moden, B., Swartz, T. A., Tirosh, M., *et al* (1983) The Arjenyattah epidemic: a mass phenomenon. *Lancet*, *ii*, 1472–1474.

Mohr, P. D. & Bond, M. J. (1982) A chronic epidemic of hysterical blackouts in a comprehensive school. *British Medical Journal*, **284**, 961–962.

British Journal of Psychiatry (1990), 157, 425–427

The Depressed Elderly Living in the Community

A Follow-Up Study

O. BEN-ARIE, M. WELMAN and A. F. TEGGIN

In a 1982 community survey, 23 elderly respondents were rated as having a depressive disorder by CATEGO. Twenty survivors were followed up three and a half years later. The depression had remitted within one year in seven cases but nine were still depressed. There is a need for ongoing education of GPs about the recognition and course of depression in the elderly.

In an overview of the course and prognosis of depressive illness in old age, Stenback (1980) commented on the paucity of studies in this area. Accordingly, we followed up 23 respondents who had been given a CATEGO-tentative diagnosis (Wing *et al*, 1974) of depressive disorder in a community psychosocial survey of the elderly in 1982. In that study 150 persons had been randomly selected from approximately 11 000 non-institutionalised 'coloured' residents aged 65 years and over living in the Eastern Zone of the Cape Town city health district (Ben-Arie *et al*, 1983, 1987). (The authors do not subscribe to classification of people according to race or ethnicity – this group was chosen for study because they live in a specific area in which welfare and medical facilities were considered to be inadequate and in need of investigation.)

In that study an initial domiciliary examination was done and a variety of psychosocial variables investigated. Respondents were then physically examined, and assessed by psychiatrists and psychologists trained in the administration of the Present State Examination (PSE; Wing *et al*, 1974, 1977) and the Mini-Mental State Examination (MMSE; Folstein *et al*, 1975).

The respondents were poorly educated (81% had less than six years of schooling) and of low socio-economic status (79% were semiskilled or unskilled). In the original study 23 respondents were tentatively rated as depressed by CATEGO, of whom four were subsequently reassigned to anxiety-related categories in the light of additional clinical information (Ben-Arie *et al*, 1987). A corrected prevalence of depression of 13% ($n = 19$) was thus revealed.

Method

Three and a half years later the 150 original respondents were followed up. Every effort was made to trace all subjects in order to determine the overall mortality rate, but unfortunately available resources did not allow for PSE assessment of all surviving respondents in order to detect new cases of depression.

The respondents originally rated as depressed were visited at home and the PSE administered. They included the four whose diagnoses had been reassigned, as we wished to determine whether they had subsequently developed a depressive illness. Dementia and resultant functional impairment were assessed by the MMSE and a social performance scale. The course of the depression and psychiatric treatment received since 1982 were noted.

To overcome observer bias the PSE and MMSE findings were assessed independently by two raters who then discussed any differences and reached agreement.

In the initial study we were struck by the absence of suicidal thoughts in all but one of the depressed respondents, despite a high rate of hypochondriasis, pessimism, and alcoholism. Information about social support had been obtained, but in order to investigate the low rate of suicidal ideation further, the strength of religious conviction and its effect on attitude to suicide were specifically inquired about at follow-up.

Results

Thirty-four of the 150 respondents had died and seven were untraceable. After excluding refusals, the proportions of the various diagnostic groups who were deceased were: depression 9% ($n = 2/23$), dementia 44% ($n = 8/18$), other diagnoses 20% ($n = 2/10$), and no psychiatric disorder 19% ($n = 17/88$). These proportions are consistent with the age distribution of each diagnostic group, the depressed respondents being generally younger (61% under 70 years of age compared with 22% in the demented group and 30–50% in the remainder).

At follow-up, two of the depressed respondents had died and one was untraceable. Of the remaining 20, nine were diagnosed as clinically depressed, and two as having psychotic disorders (schizophrenia and bipolar disorder respectively). All CATEGO diagnoses were confirmed on clinical review. The respondent with bipolar illness was the only one of the four who had originally been reassigned from the depressed category found to have an affective disorder at follow-up. One respondent was assessed as being of "low intelligence", but none of the depressed respondents showed evidence of dementia.

Assessment of the course of the affective disorder showed that of the nine respondents who were depressed at follow-up, five had been depressed intermittently and two continually. The remaining two were so vague about subjective dysphoria that information about the course of their illness was regarded as unreliable. Of those no longer depressed at follow-up, depression had lasted less than one year from the time of the original assessment in seven cases. In one case it had endured less than three years, one respondent had been intermittently depressed, and in two cases the course was not known.

Only two of the 23 respondents originally rated as depressed and advised to seek psychiatric treatment had done so; one had responded well and fairly rapidly, whereas the other had dropped out of treatment and had a continuous depression to the time of follow-up. No respondent was receiving specific treatment for depression at the time of follow-up, although 13 respondents, of whom six were rated as depressed, were in regular contact with out-patient clinics for physical ailments.

In the present study, although no one was found to have suicidal ideation of a severity sufficient to rate on the PSE, two did admit to "fleeting thoughts of suicide". All respondents regarded themselves as firmly religious (17 Christian, 3 Muslim) and most were regular church attenders despite practical difficulties. Sixteen felt that suicide was sinful and against their religious convictions, while three gave family support as a reason for not contemplating suicide; one was uncertain.

Discussion

Although the sample studied is small, we believe the results to be worth reporting in view of the paucity of longitudinal community studies of depression. Just under a third of the respondents who were depressed in the 1982 study remained depressed for less than one year, and this accords with Murphy's (1983) prospective study of a group of depressed elderly treated in hospital, a third of whom recovered within one year.

Slightly less than half of our originally depressed respondents (9 of 20) were clinically depressed after three and a half years. In most the depression had been intermittent, but some manifested a continuing dysthymic disorder. Murphy (1983) similarly found that 48% of her sample had a poor outcome, albeit at one year, but Kay *et al* (1969) reported that 72% of their previous in-patients had symptoms at five to seven years. Post (1962) found mild or severe depressive impairment in just over 80% of an elderly depressed in-patient series after six years, and a similar proportion in another sample three years after discharge (Post, 1972).

Our findings indicate that a high proportion of the depressed elderly in the community have lasting symptoms, although prolonged depression appears to be more frequent among in-patients in the studies quoted (apart from Murphy's sample). This is probably because it is the more severely depressed who are likely to required admission.

The extremely low rate of suicidal ideation in the first study was confirmed in the present study. These findings were in accord with a reported low incidence of suicide in the elderly community from which the sample was drawn, with only two cases recorded in the city health district between the years 1981 and 1986 in a population estimated at 25 000. In the earlier study the absence of suicidal ideation was considered to be due to excellent social support, but the present investigation indicates that the respondents were an extremely religious group and that this influenced their attitude to suicide. Thus their strong religious beliefs may have been an additional protective factor, as postulated by Durkheim (Stenback, 1980).

The death rate in the depressed respondents was lower than that in the other diagnostic groups. This could be explained by our finding that the mortality rate in the cohort was related to age rather than to psychiatric diagnosis, and that the depressives were markedly younger than the rest of the cohort. Furthermore, dementia had been carefully excluded in the depressed respondents in the earlier study, whereas the other diagnostic groups may have contained mild cases of dementia.

It was disturbing to note that in spite of the ready availability and use of general out-patient clinics in this community (Nash & de V Meiring, 1983), none of the six depressed respondents attending these clinics were receiving antidepressant treatment at follow-up. It appears that their depression remained unrecognised by clinic doctors and that the original survey, which received wide local publicity (Ben-Arie *et al*, 1983), had had little influence in this respect.

In conclusion, it is important to note that mild depression in the elderly in the community, even if insufficiently severe to require admission, may require long-term treatment. This is particularly so when the depression is persistent or frequent or is judged clinically to be severe. It would also seem that, despite emphasis in the medical literature, this condition all too often remains undiagnosed.

Acknowledgements

We should like to acknowledge the help of Professor L. S. Gillis, Mr A. Koch, and the staff of the MRC Clinical Psychiatry Research Unit.

References

BEN-ARIE, O., SWARTZ, L., TEGGIN, A. F., *et al* (1983) The coloured elderly in Cape Town – a psychosocial, psychiatric and medical community survey: part II. Prevalence of psychiatric disorders. *South African Medical Journal*, **64**, 1056–1061.

——, —— & DICKMAN, B. J. (1987) Depression in the elderly living in the community: its presentation and features. *British Journal of Psychiatry*, **150**, 169–174.

Folstein, M. F., Folstein, S. E. & McHugh, P. R. (1975) "Mini-Mental State": a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198

Kay, D. W. K., Garside, R. F., Roy, J. R., *et al* (1969) 'Endogenous' and 'neurotic' syndromes of depression; a 5- to 7-year follow-up of 104 cases. *British Journal of Psychiatry*, **115**, 389–399.

Murphy, E. (1983) The prognosis of depression in old age. *British Journal of Psychiatry*, **142**, 111–119.

Nash, E. S. & De V Meiring, P. (1983) The coloured elderly in Cape Town – a psychosocial, psychiatric and medical community survey: part III. A survey of physical disorders and disabilities. *South African Medical Journal*, **64**, 1089–1094.

Post, F. (1962) *The Significance of Affective Symptoms in Old Age*. Maudsley Monograph no 10. London: Oxford University Press.

—— (1972) The management and nature of depressive illnesses in later life. *British Journal of Psychiatry*, **121**, 393–404.

Stenback, A. (1980) Depression and suicidal behaviour in old age. In *Handbook of Mental Health and Aging* (eds J. E. Birren & R. B. Sloane). Englewood Cliffs, New Jersey: Prentice-Hall.

Wing, J. K., Cooper, J. E. & Sartorius, N. (1974) *The Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

——, Nixon, J. M., Mann, S. A., *et al* (1977) Reliability of the PSE (Ninth Edition) used in a population study. *Psychological Medicine*, **7**, 505–516.

*O. Ben-Arie, MB, ChB, FRCPsych, *Associate Professor*; M. Welman, MA, *Scientific Officer*; A. F. Teggin, MB, BCh, MRCPsych, *Senior Lecturer, MRC Clinical Psychiatry Research Unit, Department of Psychiatry, University of Cape Town, Groote Schuur Hospital, Observatory 7925, Republic of South Africa*

*Correspondence: *Department of Psychiatry, Queen's University, Kingston, Ontario K7L 3N6, Canada*

British Journal of Psychiatry (1990), **157**, 427–430

Post-partum Psychosis in the Assir Region of Saudi Arabia

I. H. SHOEB and G. A. HASSAN

The medical notes of 91 women who had a psychiatric illness requiring admission within nine months of childbirth were studied. The incidence of post-partum psychosis was 3 per 1000 births, of which 66% were affective psychoses.

Mental disorders in the puerperium were long held to be specific entities, distinct from other mental illnesses. In his classic review of the syndrome, Pitt (1982) examined the evolution of the concept of puerperal illness and noted that the idea of a specific puerperal psychosis was discredited early in this century by many authors. The increased risk of psychotic breakdown in the puerperum is, however, well established: women are more likely to be admitted to a psychiatric hospital following delivery than at other times in their lives (Paffenbarger, 1964; Kendell *et al*, 1976).

The incidence of post-partum psychosis is generally quoted as about one admission per 1000 births, although Paugh *et al* (1963) and Jansson (1964) gave much higher figures of 3.3 and 4.6 per 1000 respectively.

In the early stages of the illness the presentation is not typical. Most authors report non-specific prodromal symptoms of lability of mood, lack of concentration, restlessness, insomnia, confusion, clouding of consciousness, irrational ideas, and disorientation. The early presentation shows ready shifting from a picture typical of one mental illness to that of another or mixture of two.

Clinical diagnoses have varied across studies. Organic syndromes were common in earlier studies, in the 1920–40s affective illnesses were thought to be the most common, as was schizophrenia or schizo-affective illness in the 1950s, but lately affective illnesses are more often reported.

Most studies of puerperal illness have come from Western countries. It is probable that puerperal psychiatric disorders are much the same in both industrial and developing countries (Cox, 1983). The aim of this study is to provide a first-hand account of puerperal illness in a community which is largely different from European populations in cultural, social, and geographical terms.

Assir Region is a vast mountainous area in the Southern Province of Saudi Arabia, with a population of 1 097 000 in 1987. There are some patients who have to travel up to 180 miles to reach psychiatric services. The Ministry of Health has established a modern medical service, provided by 223 primary health care centres (general practices) as well as 17 modern general hospitals with a total of 2 200 beds, distributed all over Assir Region. Psychiatric services, however, are provided solely by our hospital, which was opened in 1984. This is an 82-bed acute and rehabilitation centre in Abha, the capital of the province. Private health care is limited in this area.

TABLE I

Past and family history and features of current admission of 91 admitted within nine months of childbirth

	Number	*(%)*
Past history of psychiatric disturbance *related* to childbirth	39	(37)
Past history of psychiatric disturbance *unrelated* to childbirth	22	(24)
No past psychiatric history	30	(33)
Family history *related* to childbirth	7	(8)
Family history *unrelated* to childbirth	12	(13)
No family history of psychiatric disorders	72	(79)
History of *recurrent* admissions following childbirth	14	(15)
Time of admission following delivery		
within 14 days	12	(13)
within 90 days	45	(50)
after 90 days	46	(51)
Stay in hospital		
less than 14 days	42	(46)
14–29 days	31	(34)
more than one month	18	(20)
discharge against medical advice	27	(30)

Method

Diagnosis in our psychiatric hospital is based on ICD–9 (World Health Organization, 1978). There is a computerised diagnostic statistical system linked to the Central Directorate of Psychiatric Service at the Ministry of Health, in Riyadh. Methods of treatment are the same as those used in Western Europe.

Between 1 September 1986 and 31 August 1988, 1126 patients were admitted to Abha Psychiatric Hospital. The admission registration sheets of the 414 women were examined, and those who had had a baby, live or stillborn, at any time within nine months before admission were selected. There were 102 such women, and their case notes were examined in order to establish the following: final diagnosis according to ICD–9; the cluster of symptoms in the first week of admission; the course of illness during admission and through out-patient follow-up; the response to treatment; how long after childbirth the admission occurred; the patient's past and family history; the length of hospital stay; and the presence or absence of complications during delivery. In 11 cases the notes were incomplete, unclear, or thought unreliable, and these were excluded from the study.

Cases were allocated to ICD–9 categories by the two authors independently and, where there was disagreement

TABLE II

Clinical diagnosis according to ICD–9 and relevant past history for each diagnostic category

Diagnosis (ICD–9 no.)	*No.*	*(%)*	*Past history related to childbirth*				*Past history unrelated to childbirth*			
			in patient no.	*(%)*	*in family no.*	*(%)*	*in patient no.*	*(%)*	*in family no.*	*(%)*
Puerperal neurosis (300)	3	(3)	2		–		–		–	
Affective psychoses (296)	60	(66)	26	(43)	3	(5)	11	(18)	10	(17)
major depression	26	(29)	10	(39)	–		4	(15)	3	(12)
mania hypomania	19	(21)	8	(42)	1	(53)	4	(21)	4	(21)
mixed affective	15	(16)	8	(53)	2	(13)	3	(20)	3	(20)
Schizophrenic psychoses including schizoaffective (295)	14	(15)	7	(50)	1	(7)	9	(64)	2	(14)
Paranoid state (297)	1	(1)	–		–		–		–	
Organic psychoses (294)	1	(1)	–		–		–		–	
Other non-organic psychoses (298)	12	(13)	4	(33)	3	(25)	2	(17)	–	
Total	91	(100)	39	(43)	7	(8)	23	(25)	22	(24)

(seven cases) a consensus was reached (one major depressive one in the mixed affective subgroup, one schizophrenic, and four with other non-organic psychoses).

The total number of births in Assir Region was obtained from the Birth Registration Office of Assir Regional Health Authority; in the two years between 1 September 1986 and 31 August 1988 there were 30 322.

Results

The mean age of the 91 women comprising the sample was 24.5 years (range 17–40), five were primigravida, eight were in polygamous marriages, and most (80) had been referred by their relatives, rather than the medical services.

The incidence of puerperal illness in Assir Region was calculated as 91 in 30 322 births (3 per 1000), a figure very close to results obtained elsewhere.

Table I shows the past and family history and features of current admission. Table II shows the clinical diagnosis of the 91 cases and the relevant past history for each diagnostic group. Table II shows that major depression was diagnosed in 26 patients, of whom 10 had a past psychiatric history related to childbirth, whereas history unrelated to childbirth was found in only four. A similar tendency was found with the other affective psychoses.

Comparing the 60 patients with affective psychoses with the 14 schizophrenic/schizoaffective patients, we found past history unrelated to childbirth in 11 patients out of 60 in the affective group (18%) compared with nine patients (64%) in the schizophrenic/schizoaffective group.

Of our 91 patients, five had complicated deliveries (three Caesarean sections, one stillbirth, and one premature labour); two patients had twins.

Discussion

An incidence of puerperal psychosis of 3 per 1000 births, although close to results obtained by Paugh *et al* (1963) and Jansson (1964), should be treated with caution. Of our 91 patients, only 11 were medically referred and the majority were brought by relatives. It is our impression that only severe cases were brought to hospital, and an unknown number of women suffering from puerperal illness were treated by local healers or received no treatment at all. Cox (1983), in his study of post-natal illness among African women, reported that these women were more likely to seek help from a traditional healer than to visit a doctor.

Our results also show that 87% of patients were admitted after two weeks of delivery, and half were admitted after 90 days. It is quite likely that late admission occurred only when spontaneous recovery was delayed or when traditional healers had proved unsuccessful; if this is so, the true incidence is higher than that estimated above.

Affective psychosis was diagnosed in 60 patients (66%), a slightly lower proportion than previously reported, for example by Dean & Kendell (1981), who found affective psychosis in 58 patients (82%) out of their 71 Scottish women. Dean & Kendell (1981) found only one case of schizophrenia and four of schizoaffective disorder, and a further five were diagnosed as unspecified functional psychosis. Their figures for these two diagnostic categories are significantly lower than ours. However, those authors used Research Diagnostic Criteria, which differ from ICD–9 in the number of criteria to be met for a specific diagnosis.

Platz & Kendell (1988) have suggested that childbirth is capable of precipitating episodes of illness in women with only a moderate genetic or constitutional predisposition to affective disorders. In line with those authors, our data suggest that women with affective disorder have a higher risk of puerperal breakdown than at other times, while women with schizophrenic/schizoaffective disorders have a more or less equal risk of relapse following childbirth as at other times.

In our sample, it appears that multigravida admissions to psychiatric hospitals occurred more readily than primigravida admissions. It is difficult to draw any conclusion about the relative risk factor, but we suspect that an apparent readiness to bring multigravida for admission may reflect social norms. A newly married woman is likely to spend the first few years of her marriage with the extended family (her parents or her husband's parents), where there is considerable support; she is also likely to be treated when ill in a private hospital. The couple are likely to live apart from the extended family after a few years of marriage, when they have children and are becoming more independent economically. Hence we suspect that multigravida patients have less support during pregnancy and after delivery than primigravida patients, and that may account for the increased number of multigravida admissions in our sample.

Some of our results seem to differ from those of other studies. Further investigations of various aspects of post-partum illness in Assir Region, and indeed in other areas of the world, are required, and are long overdue.

References

Cox, J. L. (1983) Post-natal depression: a comparison of Scottish and African women. *Social Psychiatry*, 18, 25–28.

Dean, C. & Kendell, R. E. (1981) The symptomatology of puerperal illness. *British Journal of Psychiatry*, 139, 128–133.

Jansson, B. (1964) Psychic insufficiencies associated with childbearing. *Acta Psychiatrica Scandinavica*, 172 (suppl. 2).

Kendell, R. E., Wainwright, S., Hailey, A., *et al* (1976) The influence of childbirth on psychiatric morbidity. *Psychological Medicine*, 6, 297–302.

Paffenbarger, R. S. (1964) Epidemiological aspects of parapartum mental illness. *British Journal of Preventative Social Medicine*, 18, 189–195.

Paugh, T. F., Jerath, B. K., Schmidt, W. M., *et al* (1963) Rates of mental illness related to childbearing. *New England Journal of Medicine*, 268, 1224–1228.

Pitt, B. (1982) Depression and childbirth. In *Handbook of Affective Disorders* (ed. E. S. Paykel), pp. 361–378. Edinburgh: Churchill Livingstone.

Platz, C. & Kendell, R. E. (1988) A matched-control follow up and family study of puerperal psychoses. *British Journal of Psychiatry*, 153, 90–94.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

*Ismail Hani Shoeb, MB, BCh, MRCPsych, *formerly Consultant Psychiatrist and Chief Medical Officer, Abha Psychiatric Hospital, Abha, Assir Region, Saudi Arabia, now Barnet Clinical Lecturer, Department of Psychiatry, University College and Middlesex School of Medicine, Riding House Street, London W1N 8AA*; Gamal Ahmed Hassan, MB, BCh, MRCPsych, *formerly Consultant Psychiatrist, Abha Psychiatric Hospital, Abha, Assir Region, Saudi Arabia, now Consultant Psychiatrist, Frimley Park Hospital, Frimley, Surrey*

*Correspondence

British Journal of Psychiatry (1990), 157, 430–433

Panic Attacks in Chronic Schizophrenia

NICHOLAS ARGYLE

Of 20 patients attending a clinic for maintenance therapy of schizophrenia, seven had regular panic attacks, and these were often associated with agoraphobia and social phobia. Similar fears and avoidance in other cases were associated with paranoid ideas and negative symptoms. The relationship of panic to psychotic symptoms varied greatly. In two patients neuroleptics were associated with an increase in panic attacks.

Since its inclusion in DSM-III (American Psychiatric Association, 1980), panic disorder has been the subject of great research interest, both clinically and in the laboratory. In DSM-III-R (American Psychiatric Association, 1987) panic disorder has taken precedence over agoraphobia in the diagnostic hierarchy – reflecting the adoption by many authors of Klein's (1980) model of panic and agoraphobia. At the same time it is becoming clear that panic attacks occur in patients with all the anxiety disorders, depression, and personality disorders, and the distinction of panic disorder as a diagnostic category has been questioned (e.g. Argyle & Roth, 1989). In particular the relationship of panic disorder to depression remains a matter of dispute. DSM-III-R now allows both diagnoses to be made. Boyd (1986) describes how commonly panic disorder occurs with other diagnoses, and suggests panic attacks lead to seeking treatment in other disorders.

There has been little attention paid to any relationship between panic attacks or panic disorder and schizophrenia. Sandberg & Siris (1987) reported one chronic schizophrenic in whom panic attacks responded to alprazolam. Kahn *et al* (1988) treated seven patients with schizophrenia and panic attacks with alprazolam and found improvement in both positive and negative symptoms. Kahn *et al* (1987) also suggest panic may contribute to the pathogenesis of schizophrenic symptoms. Benzodiazepines are generally recognised as having only a minor role in the treatment of schizophrenia, where they may reduce both anxiety symptoms and psychotic symptoms in some cases (Donaldson *et al*, 1978).

This study describes the incidence and phenomenology of panic attacks and phobias in a group of 20 patients with chronic schizophrenia.

Method

Twenty consecutive patients attending an out-patient clinic for chronic maintenance treatment of schizophrenia were interviewed. All had previous in-patient admissions and the shortest time since first psychiatric episode was four years. Diagnosis was made by clinical interview and review of extensive medical notes. All patients met both ICD-9 (World Health Organization, 1978) and DSM-III-R (American Psychiatric Association, 1980) criteria for schizophrenia and had had a chronic course of illness. Seven were of paranoid type and 13 of hebephrenic or disorganised type. None had had acute psychotic exacerbations in the last three months, but eight had residual positive psychotic symptoms. Three patients were in remission, no positive or negative schizophrenic symptoms being present. All except two were on neuroleptics. Six were living with spouses or parents, four lived in sheltered or supervised accommodation, and ten lived alone. Nine were working at least part time.

All subjects were interviewed by the author using both a clinical interview and a structured interview (SCID-UP; Spitzer & Williams, 1983) to establish DSM-III-R (American Psychiatric Association, 1987) diagnoses of panic disorder and anxiety states and depressive illness. Schizophrenia was not used as an exclusion criteria, but the relation of psychotic symptoms to anxiety symptoms was noted. All subjects had at least four follow-up interviews over 12 months to record changes in their symptoms.

Results

The average age of the 20 patients was 39.9 (range 20–62) years, and 15 were men. Four men and three women reported panic attacks, with a frequency ranging from once a week to once a day (average 2.4 per week). One of these had an excessive caffeine intake, which excluded a primary diagnosis of panic disorder.

Four of these seven patients had spontaneous panic attacks, that is, not occurring in, or before entering, phobic situations, with rapid escalation to four or more symptoms within ten minutes, and at least one attack per week over the last four weeks, and therefore met full criteria for DSM-III-R panic disorder, apart from the coexistence of schizophrenia. On close questioning of these four, two had attacks precipitated by anxious cognitions associated with overvalued paranoid ideas, and one had attacks clearly precipitated by physical sensations (which were not psychotic in nature).

Three patients had panic attacks in situations related to agoraphobic fears of a typical nature – fear of being alone, unsafe, away from home. Two of these three had spontaneous attacks also; one had only situational attacks. Of the 13 patients not experiencing panic attacks, one patient had typical agoraphobic fears and four others had travel fears related to paranoid ideas.

Social avoidance was present in 13 cases. In five this was associated with negative symptoms of loss of interest and motivation; in four paranoid ideation was the apparent cause; in four typical social phobia with fear of appearing anxious and being humiliated was found. Three of this latter group had full (four or more symptoms) panic attacks in social situations, two also having spontaneous attacks and meeting criteria for panic disorder.

One patient with panic also met criteria for major depressive episode (DSM-III-R).

Follow-up and summaries

The seven cases with panic attacks are briefly summarised.

(a) A 33-year-old man, living with his parents, with residual auditory hallucinations and thought disorder, had panic attacks which were judged to be precipitated by excessive caffeine intake (10–20 cups of coffee or tea per day plus caffeine tablets). He could not be persuaded to reduce his caffeine intake and panic attacks continued. He remained socially withdrawn and agoraphobic.

(b) A 25-year-old single man with residual paranoid delusions, working as a part-time cleaner, had his antipsychotic medication increased to reduce his delusions and sleep disturbance. At the same time his panic attacks became much less frequent and the moderate agoraphobia and social withdrawal both improved.

(c) A 34-year-old delivery man living with his mother, with residual paranoid ideation and ideas of reference, unilaterally stopped his antipsychotic medication and his panic attacks stopped. After several weeks he became increasingly agitated, paranoid, and socially avoidant. On restarting his antipsychotics (fluphenazine 25 mg i.m. three-weekly), panic attacks recurred even though his paranoid ideas and agitation were reduced. Panic attacks did not respond to a change in antipsychotics and he was unwilling to take any other drugs.

(d) A 26-year-old single man working as a nursing assistant was in remission from schizophrenia but had marked social phobia with panic disorder. These responded to behavioural instruction and low-dose benzodiazepines (diazepam 2.5 mg t.d.s.).

(e) A 32-year-old unemployed lady living with her mother, whose schizophrenic illness had always been associated with anxiety, panic attacks, and social phobia, had few residual ideas of reference and overvalued ideas but marked negative symptoms – lack of confidence, indecisiveness, and lack of motivation – which were unresponsive to medication. Alprazolam (0.5 mg t.d.s.) reduced the frequency of her panics but had no effect on her behaviour.

(f) A 60-year-old single lady living alone practised severe social avoidance related to paranoid ideas and agoraphobia associated with typical ideas of collapsing or being away from safety. She was also depressed. Panic attacks occurred mainly at home, apparently precipitated by minor bodily symptoms such as dizziness, fleeting pains, or breathlessness or palpitations. Her paranoid ideas were resistant to increased doses of antipsychotics (flupenthixol 60 mg i.m. fortnightly). Panic attacks became more frequent with the increase in antipsychotics and less frequent, though not absent, with diazepam (5 mg b.d.). She did not tolerate a trial of antidepressants. She would not attempt behavioural or cognitive therapy and remained phobic of social and travel situations.

(g) A 38-year-old lady living alone, with residual paranoid ideas and infrequent auditory hallucinations, whose social avoidance was directly attributed to paranoid ideas and fluctuated with their intensity, became more psychotic and agitated when her panic attacks became more frequent. Increased antipsychotic medication reduced the frequency. Panic attacks occurred not in phobic situations but following paranoid ideation, which made her anxious.

Discussion

Panic attacks were found to be common, occurring in seven of this sample of 20 patients. Because of the overshadowing importance of psychotic symptoms earlier in the course of illness, panic and phobic phenomena may be relatively ignored by clinicians dealing with schizophrenia. For the subgroup of seven patients who had panic attacks, panic and phobic avoidance accounted for a significant part of their current distress and disability.

The relationship between panic attacks and phobic avoidance was more complex than in other patients with anxiety states, because both negative symptoms and paranoid ideas were also found as causes of social withdrawal or avoidance behaviour.

One in two cases (b and g) were panic attacks clearly associated with global severity of illness, fluctuating with levels of psychosis, sleep disturbance, and other symptoms. In both cases they were reduced by antipsychotics. Increased arousal may be the simple connection here. Arousal is known to be high in phobic anxiety and panic (Lader & Mathews, 1968) and in some cases of schizophrenia (Gruzelier & Venables, 1972), and would be reduced by antipsychotics. While there does appear to be a specific genetic predisposition to panic there are probably many non-specific factors, such as arousal, which also increase the likelihood of panic.

In one case panic was associated with a high caffeine intake which is not uncommon in schizophrenia and which is easily overlooked. The importance of caffeine as a harmful drug is probably underestimated in the general population also (Ashton, 1987).

The phenomenology of the panic attacks themselves was the same as that found in pure anxiety states, with a high number of symptoms, usually six or more, and the same spectrum of symptoms, with one exception. None of these patients had the cognition that they were going crazy, insane, or losing control, possibly because they had had previous experience of really becoming psychotic. This supports the hypothesis that the cognitive aspect of panic, which consists of catastrophic interpretation, can be modified, in this case by prior experience (Argyle, 1988). (The subjects studied by Kahn *et al* (1988) did report the fear of going crazy, which appears to have been justified as they also experienced sudden increases in psychotic symptoms with their panic attacks; the difference may be explained by their sample being actively psychotic, chronically hospitalised, and low functioning.)

A different interpretation was remembered by two patients in this sample; when they had been more psychotic they had attributed their panic attacks to interference by external forces which reinforced their paranoid delusions. This is distinct from the occurrence of panic during the anxiety associated with paranoid delusions or ideas.

The seven patients with panic attacks did report other anxiety symptoms, particularly insomnia and poor concentration, but these were also present in several of the remaining 13 patients and could well have been related to either their schizophrenia or antipsychotic medication. Depersonalisation or derealisation were present in six of the seven panic patients and five of the other 13. However, none of the 13 reported sudden or intense episodes; periods of anxiety were described by nine of these patients but these did not have the sudden onset nor large number of symptoms seen in panic attacks.

A previous history of being misdiagnosed as schizophrenic is not too uncommon in panic disorder, and may be associated with the difficulty in describing depersonalisation/derealisation, plus the extreme nature of panic anxiety. This may give rise to descriptions of symptoms which have a forcefulness reminiscent of delusional illness. Anxiety and panic symptoms may be among the presenting symptoms of a schizophrenic illness. They may be seen either as part of a more general psychophysiological disturbance or as an understandable psychological reaction to psychotic experiences.

Patients with panic disorder misdiagnosed as psychosis or who have failed to respond to other drugs may be given antipsychotics. There is no evidence to support this treatment in pure panic disorder and many patients clinically report that their illness was exacerbated by taking antipsychotics, some describing a dramatic deterioration. In this study two cases had worsening of panic with the reintroduction or increasing of antipsychotics.

This could be due to some central effects of the medication or to misinterpretation of the peripheral side-effects and possible autonomic instability. The latter seemed to be a factor in case (f).

Conclusion

These cases provide further evidence for the occurrence of panic attacks outside of pure panic disorder, with or without agoraphobia. Their relationship to the patients' other, schizophrenic, symptoms varied from running a closely parallel course to being apparently independent. It is suggested that antipsychotics may increase panic attacks in some cases.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM–III). Washington, DC: APA.

—— (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

Argyle, N. (1988) The nature of cognitions in panic disorder. *Behaviour Research and Therapy*, 26, 261–264.

—— & Roth, M. (1989) A phenomenological study of 90 patients with panic disorder. Part II. *Psychiatric Developments*, 3, 187–209.

Ashton, C. H. (1987) Caffeine and health. *British Medical Journal*, **295**, 1293–1294.

Boyd, J. H. (1986) Use of mental health services for the treatment of panic disorder. *American Journal of Psychiatry*, **143**, 1569–1574.

Donaldson, S. R., Gelenberg, A. J. & Baldessarini, R. J. (1978) The pharmacologic treatment of schizophrenia. *Schizophrenia Bulletin*, **9**, 504–527.

Gruzelier, J. H. & Venables, P. H. (1972) Skin conductance orienting activity in a heterogenous sample of schizophrenics; possible evidence of limbic dysfunction. *Journal of Nervous and Mental Disease*, **155**, 277–287.

Kahn, J. P., Drusin, R. E., Klein, D. F., *et al* (1987) Schizophrenia, panic anxiety and Alprazolam (letter). *American Journal of Psychiatry*, **144**, 527–528.

——, ——, ——, *et al* (1988) Adjunctive alprazolam for schizophrenia with panic anxiety: clinical observation and pathogenetic implications. *American Journal of Psychiatry*, **145**, 742–744.

Klein, D. F. (1980) Anxiety reconceptualised. *Comprehensive Psychiatry*, **21**, 411–427.

Lader, M. H. & Mathews, A. M. (1968) A physiological model of phobic anxiety and desensitisation. *Behaviour Research and Therapy*, **6**, 411–421.

Sandbeg, L. & Siris, S. (1987) Panic disorder in schizophrenia. *Journal of Nervous and Mental Disease*, **175**, 627–628.

Spitzer, R. L. & Williams, J. B. W. (1983) *Structured Clinical Interview for DSM–III–R, Upjohn version. 10/15/83*. New York: New York State Psychiatric Institute.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD–9). Geneva: WHO.

Nicholas Argyle, MB, BS, MRCPsych, *formerly at Cambridge University Medical School, now c/o Physiology Department, MIU, Fairfield, Iowa 52556, USA*

British Journal of Psychiatry (1990), **157**, 433–436

Schizophrenia and Marfan Syndrome

PINKHAS SIROTA, MOSHE FRYDMAN and LEA SIROTA

Five index patients and three of their first-degree relatives were affected both by schizophrenia and Marfan syndrome. Since the association appears statistically significant, the possibility of linkage disequilibrium between adjacent genes or a cytogenetic abnormality causing both disorders is suggested. These hypotheses are testable and hold promise in attempting to map the 'schizophrenia susceptibility gene' by the candidate-gene approach.

Marfan syndrome (MS) is a dominantly inherited disorder of connective tissue. Typically, the patients are tall and have an asthenic body build. Stretching of the zonula with dislocation of the lenses, dilatation of the aortic root, prolapse of the mitral valve, and often fatal dissection of the aorta are the most characteristic and serious findings. Many additional features have been reported, but in the absence of a biochemical test or marker, the presence of at least one major musculoskeletal, cardiac or ocular feature, in addition to the typical phenotype, is necessary for unequivocal diagnosis (Pyeritz & McKusick, 1979).

A patient with MS and schizophrenia has been reported by Romano & Linares (1987). We report five unrelated patients and three of their first-degree relatives, who had both conditions.

Case reports

Patients 1–4 were in-patients of Gehah Psychiatric Hospital and were referred and admitted for schizophrenia. In patients 2 and 3, MS was suspected clinically by one of us (PS) and the diagnosis confirmed as indicated in Table I. Cases 1 and 4 were ascertained by systematically studying patient records, looking for MS. Patient 5 was referred from a genetic clinic in another hospital because of suspected schizophrenia. Information regarding three additional cases was obtained through family studies of the index patients. The family pedigrees are shown in Fig. 1.

The diagnosis of MS in our patients complied with the stringent criteria of Pyeritz & McKusick (1979). Similarly, the diagnosis of schizophrenia was based on the DSM–III–R classification (American Psychiatric Association, 1987).

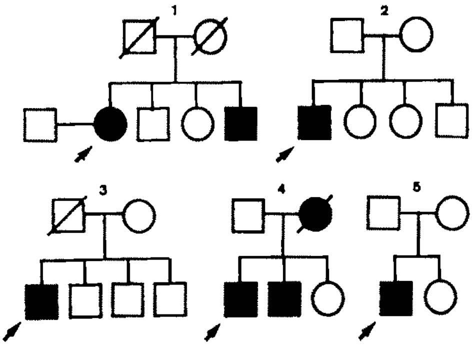


FIG. 1. Family pedigrees 1–5, indicated by arrows (□ male, ○ female, filled shapes indicate sufferers and bars indicate deceased family members).

Case 1

The patient was evaluated at the age of 51 years. She has a brother with schizophrenia and MS, a sister with hyperglycosuria who is otherwise normal, and an unaffected brother. The patient is of Ashkenazi Jewish origin. Her

TABLE I

Characteristics of five patients with the Marfan syndrome and schizophrenia

	1	*2*	*3*	*4*	*5*
Sex	F	M	M	M	M
Age: years	51	19	26	23	19
Height: m (percentile)	1.78 (>97)	1.98 (>97)	1.97 (>97)	2.01 (>97)	1.86 (90)
Clinical features					
Ocular					
ectopia lentis	+	–	–	+	–
myopia	–	+	+	–	+
other	+	–	–	–	–
Cardiovascular					
clinical evidence for mitral-valve prolapse	+	+	+	+	+
echo evidence for mitral-valve prolapse	not done	+	+	–	+
clinical evidence for aortic regurgitation	+	+	+	–	+
echo evidence for aortic regurgitation	not done	+	+	–	+
Musculoskeletal					
arachnodactyly	+	+	+	+	+
pectus deformity	+	+	+	+	+
high narrow palate	+	+	+	+	+
joint hypermobility	+	+	+	+	+
vertebral column deformity	+	+	+	+	+
inguinal hernia	–	+	–	+	–
Positive family history	MS/S[1]	-	-	MS/S[1]	-
Age at onset of schizophrenia: years	19	14	19	20	17
Type of schizophrenia	Paranoid	Disorganised	Disorganised	Paranoid	Paranoid

[1] Marfan syndrome and schizophrenia.

parents and the rest of her family perished in the Holocaust and no other data are available. The diagnosis of MS was based on the findings reported in Table I, including cataract and adhesions of the lens to the cornea.

She was admitted several times for long periods in different psychiatric hospitals because of thought disorders with persecutory delusions, ideas of reference, blunted affect, and auditory hallucinations, with constant deterioration in her personality and narrowing of her ability to form interpersonal relationships.

Case 2

A 19-year-old male of Sephardic Jewish origin had multiple admissions to different mental hospitals because of schizophrenia since the age of 14. The diagnosis of MS was based on skeletal, ocular and cardiovascular findings (Table I). In addition, the metacarpal index of the second and third metacarpals in both hands was 9.4 and 9.2 respectively (normal up to 8.7).

His parents and three siblings are normal.

Case 3

A 26-year-old male of Ashkenazi Jewish origin had, since 14 years of age, presented disturbed emotional and social behaviour, and at 19 years of age he had an acute psychotic episode which led to admission and to his premature discharge from military service. He was diagnosed as suffering from hebephrenic schizophrenia (disorganised type).

The diagnosis of MS was established at 26 years of age and was based on the typical musculoskeletal, ocular and cardiac findings (Table I).

His parents and three siblings are normal.

Case 4

A 23-year-old male of Sephardic Jewish origin was found to have schizophrenia of the paranoid type. The diagnosis of MS was based on the typical musculoskeletal ocular and cardiac features (Table I).

His mother, a schizophrenic, died of heart disease associated with MS and an affected brother had cardiac surgery and is reported to suffer from schizophrenia. The father and another sibling are normal.

Case 5

A 19-year-old male of Sephardic Jewish origin was admitted to a psychiatric hospital at age 17 because of a paranoid hallucinatory episode. Subsequently, he had multiple psychiatric admissions and was diagnosed as having schizophrenia, paranoid type.

The patient had the typical musculoskeletal, ocular and cardiac findings of MS (Table I).

The parents and his sister are normal.

Laboratory studies

Urinary excretion of amino acids was normal in all patients and homocystinuria and cystinuria were excluded by the cyanide nitroprusside test.

Discussion

Although the basic defect in MS is yet unknown, many lines of evidence indicate that abnormal collagen is being produced (Pyeritz & McKusick, 1981). In fact, a 38-base-pair insertion in the gene encoding the alpha-2 chain of type I collagen on chromosome 7q was reported in one case by Henke *et al* (1985). However, other workers did not find linkage between MS and the four major fibrillar collagen genes on chromosomes 17q, 7q, 12q, and 2q (Ogilvie, *et al*, 1987). These data support the notion that MS is genetically heterogeneous, and raise the possibility that a mutation in a gene responsible for post-transcriptional modification of collagen is the cause of MS (Pyeritz & McKusick, 1981; Ogilvie *et al*, 1987).

Sherrington *et al* (1988) studied five Icelandic and two British families and found strong evidence localising a schizophrenia-susceptibility locus to proximal 5q ($z = 6.49$). However, another group of investigators studying the same markers and several other loci on the long arm of chromosome 5 found no suggestion of linkage with any of the markers studied. Furthermore, they were able to exclude linkage across 40 map units (cM) of this region (Kennedy *et al*, 1988). Since schizophrenia is probably genetically hetereogeneous, these results are not necessarily contradictory (Lander, 1988).

In order to estimate the possible significance of the concomitant occurrence of schizophrenia and MS in our patients, we assumed that the number of individuals with both disorders follows a binominal distribution. Since the sample size was very large (Jewish population 3.436×10^6), we used a normal approximation. Classic MS is relatively rare, with a prevalence of 4–6/100 000 (Pyeritz & McKusick, 1979). Schizophrenia is more common, with a lifetime prevalence 2–4/1000 (Kaplan & Sadock, 1985). The joint probability of independently acquiring both conditions is 8×10^{-8} to 24×10^{-8}. Our observation of five index patients gives a population frequency of 6.872×10^{-5}, which exceeds the 95th percentile (9.957×10^{-7}), under the assumption of independence of the two conditions.

Although it can be argued that the prevalence of MS, schizophrenia, or both is greater in Israel than in other parts of the world, there is no evidence to support such a view. Admittedly, ascertainment bias could account for the observation of MS among schizophrenic patients; however, since four of the cases were found among the patient population of a single mental hospital, and since we did not include the three additional familial cases in the calculations, we feel that our estimate may be conservative.

It is possible that a factor related to MS, such as social isolation, the constant threat of the potentially fatal disease, and the emotional turmoil that accompanies the diagnosis, reveal disease symptoms which otherwise would not have been expressed. Nevertheless, the fact that schizophrenia was already established when MS was diagnosed in patients 2 and 3 does not favour this hypothesis.

In the two families (1, 4) in which MS occurred more than once, the affected relatives also had schizophrenia, while non-affected relatives had neither. This observation and the seemingly non-random association between MS and schizophrenia may result from linkage disequilibrium between the MS gene and the heritable component of schizophrenia. A minute chromosomal aberration (deletion, trisomy or translocation) involving two adjacent genes may be responsible for such a phenomenon. This possibility may explain the presumed *de novo* occurrence of schizophrenia and MS in patients 2, 3 and 5. It is interesting to note in this respect that Bassett *et al* (1988) reported an uncle and nephew with schizophrenia and partial trisomy of chromosomal band 5q11.2-5q13.3. Once the assignment of the MS gene is established, this possibility may become testable by looking for linkage between schizophrenia and polymorphic genetic markers located close to the MS locus.

Finally, as argued above, both schizophrenia and MS are probably genetically heterogeneous, and so several conditions with a similar phenotype may each be determined by a mutation in a different gene, with a different chromosomal location. It is conceivable that a schizophrenic condition may be part of the phenotype of a Marfan-like syndrome, in which case the association reflects a pleitropic effect of the mutant gene itself (i.e. one gene with multiple effects). Such a possibility may explain on ethnic grounds both the rarity of this association and its peculiar occurrence in Israel.

Acknowledgements

We would like to thank Professor Yosi Hochberg, Statistical Laboratory, Tel Aviv University, for the statistical analysis, Professor H. Munitz, Gehah Psychiatric Hospital, for his help, and Professor Richard M. Goodman, Sheba Medical Center, for referring patient 5.

References

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III – R). Washington, DC: APA.

Bassett, A. S., McGillivray, B. C., Jones, B. D., *et al* (1988) Partial trisomy chromosome 5 cosegregrating with schizophrenia. *Lancet*, *i*, 799–801.

Henke, E., Leader, M., Tajima, S., *et al* (1985) A 38 base pair insertion in the pro alpha 2 (1) collagen gene of a patient with Marfan syndrome. *Journal of Cellular Biochemistry*, **27**, 168–174.

Kaplan, H. I. & Sadock, B. J. (eds) (1985) *Comprehensive Textbook of Psychiatry* (4th edn), p. 644. Baltimore: Williams and Wilkins.

Kennedy, J. L., Giuffra, L. A., Moises, H. W., *et al* (1988) Evidence against linkage of schizophrenia to markers on chromosome 5 in a northern Swedish pedigree. *Nature*, 336, 167–170.

Lander, E. S. (1988) Splitting schizophrenia. *Nature*, 336, 105–106.

Ogilvie, D. J., Wordsworth, B. P., Priestley, L. M., *et al* (1987) Segregation of all four major fibrillar collagen genes in the Marfan syndrome. *American Journal of Human Genetics*, **41**, 1071–1082.

Pyeritz, R. E. & McKusick, V. A. (1979) The Marfan syndrome: diagnosis and management. *New England Journal of Medicine*, **300**, 772–777.

—— & —— (1981) Basic defects in the Marfan syndrome. *New England Journal of Medicine*, **305**, 1011–1012.

Romano, J. & Linares, R. L. (1987) Marfan syndrome and schizophrenia: a case report (letter). *Archives of General Psychiatry*, **44**, 190–192.

Sherrington, R., Brynjolfsson, J., Petursson, H., *et al* (1988) Localisation of a susceptibility locus for schizophrenia on chromosome 5. *Nature*, **336**, 164–167.

*P. Sirota, *Head of Department of Psychiatry, Yehuda Arbarbanel Mental Health Center Bat Yam, Israel,* and *Sackler School of Medicine, Tel Aviv University, Israel*; M. Frydman, *Senior Lecturer in Pediatrics, Genetics Clinic, Department of Pediatrics, Golda Medical Center, Hasharon Hospital, Petah Tiqwa and Sackler School of Medicine, Tel Aviv University, Israel*; L. Sirota, *Senior Neonatologist, Neonatology Department Golda Medical Center, Hasharon Hospital, Petah Tiqwa and Sackler School of Medicine, Tel Aviv University, Israel*

*Correspondence: *Yehuda Abarbanel Mental Health Center, Bat Yam, Israel*

British Journal of Psychiatry (1990), **157**, 437–438

Carbamazepine and Forme Fruste Neuroleptic Malignant Syndrome

TIM DALKIN and ALAN S. LEE

A woman developed rigidity, autonomic instability and altered consciousness after taking an overdose of trifluoperazine and carbamazepine. A diagnosis of NMS was made despite the absence of fever, as carbamazepine might modify the presentation of NMS.

Neuroleptic malignant syndrome (NMS) is characterised by fever, rigidity, autonomic instability, and alteration of consciousness following exposure to neuroleptics (Caroff, 1980; Levenson, 1985). It is usually associated with elevation of creatine phosphokinase (CPK) levels and a leukocytosis. We report a patient who developed generalised rigidity, autonomic instability, and impairment of consciousness in the absence of fever, following an overdose of trifluoperazine and carbamazepine.

Case report

A 58-year-old married woman was admitted comatose to the acute medical wards of a teaching hospital. She was well known to psychiatric services, as she had a 27-year history of bipolar affective disorder. She had been admitted on 17 occasions in both manic and depressive episodes and had taken two previous overdoses. At the age of 21 she had developed nocturnal epilepsy which had been treated with primidone. She had subsequently been free of fits for 30 years. Before this admission she had been taking chlorpromazine (200 mg q.d.s.), trifluoperazine (10 mg t.d.s.), procyclidine (5 mg t.d.s.), and carbamazepine (400 mg b.d.; serum levels 7–8 mg/l).

She was admitted after taking an impulsive overdose of an unknown quantity of trifluoperazine and carbamazepine, which she immediately disclosed to her husband. On arrival at hospital two hours later she was in stage-3 coma. Her gag reflex was impaired, allowing the passage of an endotracheal tube without resistance, and she had bilateral extensor plantar reflexes. She was monitored overnight on the intensive care unit before being transferred to a medical ward, where she was reported to be drowsy and disorientated. She felt hot and requested a bedside fan, although her maximum recorded temperature was only 37.2 °C.

Two days after the overdose she was reviewed by one of the authors (TD) when the following signs were noted: generalised muscular rigidity, involuntary jerking movements in her face and limbs, diaphoresis, and tachycardia (120 beats/min). Her blood pressure over the previous 24 hours had fluctuated between 150/100 and 100/70 mmHg. She was disorientated in time. She was apyrexial.

Serum analysis revealed elevated CPK at 1710 IU/l (normal range up to 200 IU/l) but no leukocytosis. Renal function and liver function were normal.

All neuroleptic medication was withheld and she improved with simple supportive measures. Three days later she was fit for transfer to the psychiatric unit. She was fully orientated and apyrexial. Muscle tone was initially normal but the following day she developed cogwheel rigidity which responded to procyclidine. Her pulse was 80/min, blood pressure 140/80 mmHg, and her plantar reflexes were flexor. One week later her serum CPK had fallen to 79 IU/l. Her mental state fluctuated rapidly from sadness and self-reproach to overexcitement and irritability. Further neuroleptics were felt to be contraindicated so carbamazepine was restarted as sole treatment. Over the following week her mood stabilised and she was discharged two weeks later entirely well.

Discussion

Neuroleptic malignant syndrome typically disturbs four areas of function: temperature regulation, muscle tone, the autonomic nervous system, and level of consciousness. However, the status of partial forms remains controversial.

Levenson (1985) suggested three major and six minor criteria for diagnosing NMS, requiring all three major, or two major and four minor criteria. His major criteria are fever, rigidity, and elevated CPK, while minor criteria are tachycardia, abnormal blood pressure, tachypnoea, altered consciousness, diaphoresis, and leukocytosis. These guidelines underwrite a spectrum concept of NMS and Levenson suggests further case reports are needed. The case we describe satisfies two major and four minor criteria, and follows an overdose of trifluoperazine.

Since the diagnosis of NMS in this case is dependent on the elevation of serum CPK, other causes of this should be considered. One possibility is that since carbamazepine in overdose can induce seizures, our patient may have shown a raised CPK level following a convulsion. However, reports from her husband and nursing staff allow us to exclude this. A second possibility is that neuroleptic-induced extrapyramidal side-effects, in particular severe dystonic reactions, might themselves raise CPK levels, independent of NMS. But there are no reports in the literature of neuroleptic-induced dystonic reactions causing raised CPK levels. A third possibility is that the overdose of carbamazepine could be responsible. There is one case report of elevation of serum CPK following a carbamazepine overdose (Bursztyn *et al*, 1987). This was associated with ataxia and nystagmus, but in that report no features of NMS were present. The CPK did not reach the extremely elevated level seen in our patient. We believe, therefore, that our patient satisfied Levenson's guidelines and that the elevated CPK may be attributed to a variant of NMS.

Three other case reports are of particular interest. Sullivan (1987) described rigidity, autonomic instability, altered consciousness, and elevated serum CPK levels in the absence of fever in a man treated with zuclopenthixol decanoate and carbamazepine. Müller *et al* (1988) described NMS with mild fever (37.8 °C) in a man treated with clozapine and carbamazepine. They suggested that as clozapine does not cause extra pyramidal side-effects it would not be expected to cause NMS, and that carbamazepine may have facilitated its development. Finally, Goldwasser *et al* (1989) reported a man with previous symptoms suggesting NMS (without fever) who developed classic NMS when treated with thioridazine. Thioridazine was discontinued and the symptoms began to resolve. Carbamazepine was commenced as sole treatment six days later. Within 24 hours the patient developed fever, fluctuating blood pressure, and elevated CPK levels, which again resolved when carbamazepine was withdrawn.

The pathophysiology of NMS is unclear, but the most popular hypothesis is of excessive dopamine-receptor blockade in the basal ganglia producing extrapyramidal symptoms, and in the hypothalamus producing fever and autonomic disturbance (Szabadi, 1984). Carbamazepine is not known to affect dopaminergic systems. It is a noradrenaline reuptake inhibitor, which probably accounts for its ability to produce dyskinesias only rarely (Evans & Gualtieri, 1985). In common with lithium it inhibits limbic kindling, reduces GABA turnover, and inhibits accumulation of cyclic adenosine monophosphate (Birkhimer *et al*, 1985). Keck *et al* (1987) have shown that there is an increased rate of NMS in those patients treated with a combination of neuroleptics and lithium. The reports of Müller and Goldwasser suggest that in conjunction with neuroleptics, carbamazepine may also facilitate the development of NMS. Perhaps the neurochemical actions it shares with lithium are responsible for this. Furthermore, we wonder if carbamazepine may modify the usual presentation of NMS, increasing the likelihood of apyrexial forms.

Adityanjee *et al* (1988) suggested that NMS should be diagnosed only in the presence of fever over 39 °C, generalised rigidity, autonomic disturbance, and altered consciousness, with raised serum CPK levels having only a supporting role. We believe that this may cause the diagnosis of NMS to be missed, with important implications for treatment. Until more is known about the pathophysiology of NMS, and further cases are reported, Levenson's guidelines would appear to have greater clinical and heuristic value.

References

Adityanjee, Singh, S., Singh, G., *et al* (1988) Spectrum concept of neuroleptic malignant syndrome. *British Journal of Psychiatry*, **153**, 107–111.

Birkhimer, L. J., Curtis, J. L. & Jann, M. W. (1985) Use of carbamazepine in psychiatric disorders. *Clinical Pharmacy*, **4**, 423–434.

Bursztyn, M., Nussinovich, N. & Rosenthal, T. (1987) Muscle enzyme elevation in carbamazepine intoxication. *Israeli Journal of Medical Science*, **23**, 905.

Caroff, S. N. (1980) The neuroleptic malignant syndrome. *Journal of Clinical Psychiatry*, **41**, 79–83.

Evans, R. W. & Gualtieri, C. T. (1985) Carbamazepine: a neuropsychological and psychiatric profile. *Clinical Neuropharmacology*, **8**, 221–241.

Goldwasser, H. D., Hooper, J. F. & Spears, N. M (1989) Concomitant treatment of neuroleptic malignant syndrome and psychosis. *British Journal of Psychiatry*, **154**, 102–104.

Keck, P. E., Pope, H. G. & McElroy, S L. (1987) Frequency and presentation of neuroleptic malignant syndrome: a prospective study. *American Journal of Psychiatry*, **144**, 1344–1346.

Levenson, J. L. (1985) Neuroleptic malignant syndrome. *American Journal of Psychiatry*, **142**, 1137–1145.

Muller, T., Becker, T. & Fritze, J. (1988) Neuroleptic malignant syndrome after clozapine plus carbamazepine. *Lancet*, *ii*, 1500.

Sullivan, C. F. (1987) A possible variant of the neuroleptic malignant syndrome. *British Journal of Psychiatry*, **151**, 689–690.

Szabadi, E. (1984) Neuroleptic malignant syndrome. *British Medical Journal*, **288**, 1399–1400.

*Tim Dalkin, MB, BS, MRCPsych, *Registrar, Department of Psychiatry, B Floor, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH*; Alan S. Lee, MA, MRCPsych, *Consultant Psychiatrist and Clinical Teacher, University Hospital, Nottingham*

*Correspondence

British Journal of Psychiatry (1990), **157**, 439–441

Klüver–Bucy Syndrome and Psychiatric Illness

D. J. CLARKE and N. S. BROWN

A 52-year-old woman, whose initial psychiatric presentation at the age of 15 was with a disorder resembling schizophrenia, developed symptoms of Klüver–Bucy syndrome, and is now thought to suffer from an organic psychotic disorder. Klüver–Bucy syndrome must be distinguished from symptoms of schizophrenia or affective disorder; its presence suggests an organic process.

Klüver & Bucy (1939) described a syndrome which followed bilateral removal of the temporal lobes of rhesus monkeys, and which consisted of 'psychic blindness' (inability to recognise familiar objects, even after repeated exposure), 'hypermetamorphosis' (a strong tendency to attend and react to every visual stimulus), oral exploration of available objects, emotional unresponsiveness, and an increase in sexual activity. A similar syndrome was reported in man by Terzian & Dalle Ore (1955), who described a 19-year-old man with epilepsy who underwent bilateral anterior temporal lobectomy with removal of most of the uncus and hippocampus. This patient was unable to recognise close relatives. He displayed an increase in sexual activity, emotional unresponsiveness, increased appetite, and a severe memory disturbance, but did not show oral exploratory behaviour. Further patients with elements of the Klüver–Bucy syndrome have been described in association with Alzheimer's disease, Pick's disease and encephalitis (Lilly *et al*, 1983), adrenoleukodystrophy (Powers *et al*, 1980), trauma (Lilly *et al*, 1983; Stewart, 1985), toxoplasmosis and hypoglycaemia (Poeck, 1969), acute intermittent porphyria (Guidotti *et al*, 1979), Huntington's disease (Janati, 1985), and vascular lesions (Poeck, 1969; Shraberg & Weisberg, 1978).

When Klüver–Bucy syndrome occurs in man it may be associated with a mixed anterograde–retrograde amnesia; with verbal, rather than physical, hypersexuality; with manual, rather than oral, exploratory behaviour; and with rage reactions (Shraberg & Weisberg, 1978; Stewart, 1985). Rage reactions and some other features of Klüver–Bucy syndrome in man may respond to treatment with carbamazepine (Stewart, 1985).

Psychiatrists unfamiliar with Klüver–Bucy syndrome may fail to distinguish the features from symptoms of schizophrenia or affective disorder. The importance of the syndrome lies in its association with organic disorders.

Case report

M was born in 1936 after an uneventful pregnancy and delivery. Her mother was a 26-year-old housewife, her father a 26-year-old factory worker. She attended school from the age of five and was described in contemporary reports as an average scholar. She left school at the age of 15 to start manual work with a large industrial company, but gave the job up after a few months because she had difficulty concentrating and felt tired. She complained of a peculiar sensation, which she described as a cloud settling on her, and told her parents that she could hear voices in her head.

In 1951 she was admitted to a psychiatric hospital and was noted to be pyrexic. She was treated with electroconvulsive therapy (ECT), improved, and was discharged after three weeks. At home, however, she rapidly became withdrawn and silent, and would sit for hours hardly moving and not replying to questions. At other times she had difficulty keeping still and would bite her nails continually. She giggled to herself, refused to wash, and insisted on sleeping in her clothes.

In 1952 (aged 16) she was admitted to a different psychiatric hospital, and at this time was incontinent of urine and faeces. A diagnosis of hebephrenic schizophrenia was made, and insulin treatment started. She had a total of 30 comas lasting 5–20 min, induced by doses of insulin of up to 260 units daily. She had tonic–clonic seizures on 18 occasions. There was no improvement. By March 1953 she was extremely restless, was emotionally incongruous, and talked to herself as if hallucinating. Over the following months she became mute and then developed elements of the Klüver–Bucy syndrome. She continually explored objects with her hands and mouth, ripped fabric from furnishings and ate it, ate her faeces, removed her clothes, and was often physically aggressive for no apparent reason. She also giggled, clucked and grimaced continually, and although she did not speak, could sing simple songs if prompted. M was nursed in a high-dependency unit. Treatment with reserpine, pyridoxine, nicotinic acid, ECT, perphenazine, thioridazine, and trifluoperazine produced no improvement. Between 1969 (aged 33) and 1971 (aged 35) she had four spontaneous tonic–clonic seizures.

Her distress and her behavioural abnormalities were so great that after discussion with, and consent from, her father, and with the agreement of an independent psychiatrist, she was referred for psychosurgery.

Pre-operatively electroencephalography (EEG) showed high-voltage sharp and slow waves appearing synchronously between the frontal bases and the parietal convexities on both sides.

A posterior cingulectomy and cortical biopsy was performed in 1974, when M was 38. The biopsy contained normal brain tissue. A paramedian frontal lobotomy was performed after an interval of three months. M improved after this operation, becoming less agitated and aggressive, although with a tendency to become more disturbed premenstrually. In 1985, at the age of 49, M developed an intestinal obstruction. A colonic mass was removed and a colectomy and hysterectomy were performed. A specimen of the mass sent for histological examination showed lymphocytic lymphoma, with involvement of lymph nodes but with cut ends free of tumour. M was referred to a haematologist, who felt no further action was necessary. Since the operation she has had occasional episodes of constipation, but no weight loss or other signs of malignancy.

M continues to be nursed on a high-dependency unit. Her activity on the ward is minimal and largely purposeless. She is occasionally physically aggressive, usually in response to provocation by other disturbed residents. Her appetite is reasonable and she is able to feed herself. She dresses herself with assistance. Her speech consists of a few words, which are poorly enunciated and sometimes used inappropriately. She appears to be disorientated in time and place, and needs to be escorted when walking within the hospital. M is visited regularly by her father, but she shows no anticipation of the visits or any recognition of her father or other relatives. On one occasion she needed regular intravenous injections, and when she saw the doctor approaching became agitated and shouted 'needle, needle'. She is incontinent of urine and faeces, and has an orofacial dyskinesia but no other abnormal movements. She has no gross motor or sensory deficits, and although her tendon reflexes are bilaterally brisk, they are not pathologically so. EEG performed in 1988 only showed abnormalities consistent with psychosurgery, and a computerised tomography scan of the head showed a minor degree of cortical atrophy in the parietal lobes, and frontal-lobe atrophy presumed to be a result of the leucotomy. No temporal-lobe abnormalities were seen.

In view of the Klüver–Bucy symptoms, seizures, incontinence, disorientation, amnesia, and aphasia, the presumptive diagnosis is now of an organic psychotic disorder of unknown aetiology.

Discussion

M's initial presentation was with symptoms suggestive of schizophrenia. The notes document disordered speech, thought blocking, and catatonic symptoms. The underlying basis for the psychotic symptoms may have been an encephalitis. Psychiatric symptoms were preceded by tiredness and lethargy, and she was pyrexic on first admission. Mahendra (1981) noted a decline in catatonic symptoms since the 1930s, and postulated the decline of an infectious illness as a possible cause. The normal brain biopsy, however, argues against an encephalitic aetiology. An alternative explanation is that treatment of an initial schizophrenic illness with deep insulin therapy produced a chronic organic psychotic syndrome (dementia) as a result of prolonged hypoglycaemia. Dementia was a recognised complication of insulin treatment (Spencer, 1948). However, the notes do not document a prolonged coma or a sudden deterioration in M's condition at any point during treatment with insulin.

Her present clinical state strongly suggests that the end result of whatever morbid process occurred has been an organic psychotic disorder (dementia) of unknown origin. The diagnosis could have been revised earlier if the Klüver–Bucy symptoms had been recognised, and a more accurate prognosis provided for the patient's relatives.

If several of the features of Klüver–Bucy syndrome occur in a patient with an apparently uncomplicated affective or schizophrenic illness, the psychiatrist should be alerted to the probability of pathology of temporal-lobe limbic structures. Symptoms identical to those in affective and schizophrenic psychoses may be produced by pathology in this area (Malamud, 1967). Psychiatric illness with low mood, delusions and hallucinations may precede the Klüver–Bucy syndrome when it occurs in association with Alzheimer's or Pick's disease (Pilleri, 1966). The presence of Klüver–Bucy symptoms should therefore suggest that any accompanying psychotic symptoms have an underlying organic cause.

Acknowledgements

We are grateful to the late Dr B. H. Burns for his encouragement to report the case, and to Dr T. A. Betts who drew attention to the Klüver–Bucy symptoms and whose comments on the manuscript were most helpful.

References

GUIDOTTI, T. L., CHARNESS, M. E. & LAMON, J. M. (1979) Acute intermittent porphyria and the Klüver–Bucy syndrome. *Johns Hopkins Medical Journal*, **145**, 233–235.

JANATI, A. (1985) Kluver–Bucy syndrome in Huntington's chorea. *Journal of Nervous and Mental Disease*, **173**, 632–635.

KLÜVER, H. & BUCY, P. C. (1939) Preliminary analysis of functions of the temporal lobes in monkeys. *Archives of Neurology and Psychiatry*, **42**, 979–1000.

LILLY, R., CUMMINGS, J. L., BENSON, D. F., *et al* (1983) The human Kluver–Bucy syndrome. *Neurology (Cleveland)*, **33**, 1141–1145.

MAHENDRA, B. (1981) Where have all the catatonics gone? *Psychological Medicine*, **11**, 669–671.

MALAMUD, N. (1967) Psychiatric disorder with intracranial tumors of limbic system. *Archives of Neurology*, **17**, 113–123.

PILLERI, G. (1966) The Klüver–Bucy syndrome in man. A clinico-anatomical contribution to the function of the medial temporal lobe structures. *Psychiatria et Neurologia (Basel)*, **152**, 65–103.

POECK, K. (1969) Pathophysiology of emotional disorders associated with brain damage. In *Handbook of Clinical Neurology, Vol. 3* (eds P. J. Vinken & G. W. Bruyn). Amsterdam: North-Holland Publishing.

Powers, J. M., Schaumburg, H. H. & Gaffney, C. L. (1980) Klüver-Bucy syndrome caused by adreno-leukodystrophy. *Neurology (Ny)*, **30**, 1231-1232.

Shraberg, D. & Weisberg, L. (1978) The Klüver-Bucy syndrome in man. *Journal of Nervous and Mental Disease*, **166**, 130-134.

Spencer, A. M. (1948) Post-hypoglycaemic encephalopathy in Sakel's insulin treatment. *Journal of Mental Science*, **94**, 513-554.

Stewart, J. T. (1985) Carbamazepine treatment of a patient with Klüver-Bucy syndrome. *Journal of Clinical Psychiatry*, **46**, 496-497.

Terzian, H. & Dalle Ore, G. (1955) Syndrome of Klüver and Bucy reproduced in man by bilateral removal of the temporal lobes. *Neurology (Minneap)*, **5**, 373-380.

*D. J. Clarke, MRCPsych, *Clinical Lecturer, University of Birmingham, Department of Psychiatry, Queen Elizabeth Hospital, Birmingham B15 2TH*; N. S. Brown, MRCPsych, *Consultant Psychiatrist, Hollymoor Hospital, Northfield, Birmingham B31 5EX*

*Correspondence

British Journal of Psychiatry (1990), **157**, 441-444

Mood Disorder, 'Pre-ictal' Psychosis and Temporal Lobe Damage

BANKOLE A. JOHNSON and LACHLAN B. CAMPBELL

A patient with an abnormality in the right temporal lobe presented with episodes of mania many years before the clinical manifestation of both a simple partial seizure and complex partial seizures.

An association between epilepsy and psychotic illness is well recognised. Falret (1854) was one of the first to suggest such an association which was further supported by Slater & Beard (1963) in their description of psychosis developing on average 14.1 years following the diagnosis of epilepsy. Flor-Henry (1969) linked schizophreniform and manic-depressive psychosis to abnormalities of the dominant and non-dominant temporal lobes respectively. Therefore, the development of psychosis following epilepsy has been associated with the duration of epilepsy, abnormality of the temporal lobe itself, and with brain damage. We report a novel case of abnormality of the right temporal lobe, with episodes of mania many years before the clinical manifestation of both a simple partial seizure and complex partial seizures.

Case report

The patient was a 19-year-old man brought to the UK from Africa at the age of one. There was no family history of mental illness or epilepsy, and his early developmental milestones and obstetric history were within normal limits. However, he was later ascertained as being in need of special education and subsequently attended a residential school for children with moderate learning difficulties. His premorbid personality was described as shy and friendly.

At the age of 15, following a sexual assault, he became overactive, distractible and destructive, and was admitted in quick succession to two different psychiatric hospitals. No specific diagnosis was made on either occasion. His condition settled rapidly and he was placed in a local children's home. The following year he again suffered a relapse in his mental condition and was admitted to yet another psychiatric hospital, where a diagnosis of hypomania was made. His mood settled rapidly and he was discharged to a local hostel on no medication.

At the age of 18, he suddenly was unable to sleep for more than three hours at night and again became overactive, distractible, irritable, and impulsive. There were no reports suggestive of delusions or hallucinations. After three years in this state he ran into the street carrying a billiard cue and attacked a car and its driver. He was arrested and placed in a local remand centre. There, on the basis of a full mental state examination (including cognitive assessment), a diagnosis of hypomania was made, and he was admitted to a secure unit. The patient settled after a few days on regular sodium amytal medication, and lithium carbonate

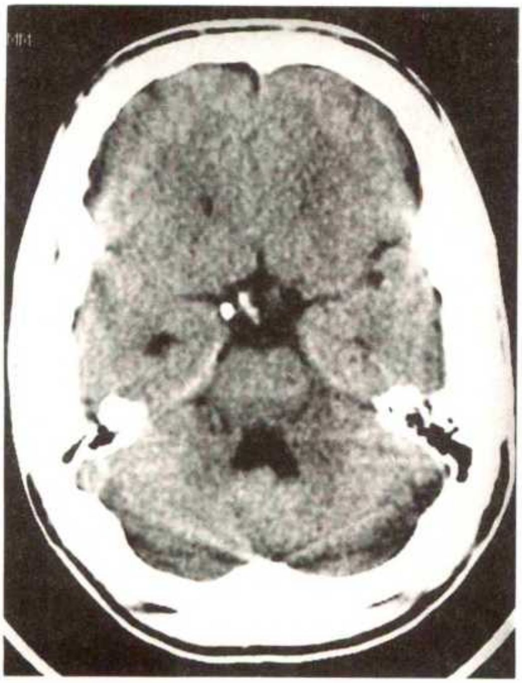


FIG. 1. CT scan illustrating right temporal horn ventricular dilatation.

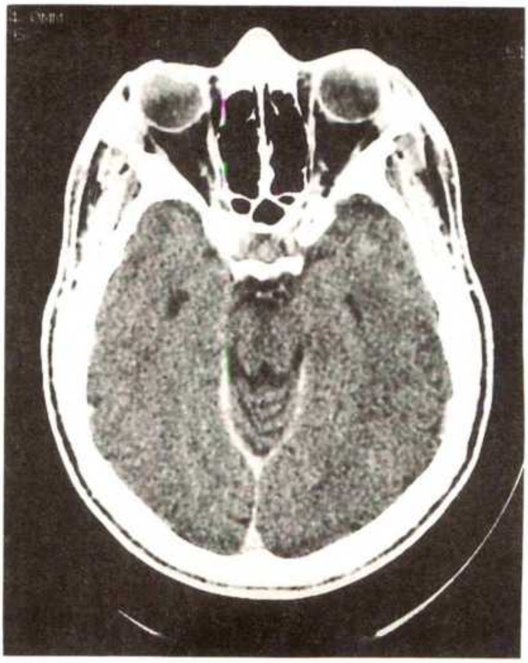


FIG. 2. CT scan showing cortical atrophy and possible sclerosis in Ammon's horn.

was then commenced. His mental state remained stable on therapeutic levels of lithium carbonate (0.8 mmol/l) and day visits to his hostel were then instigated.

During an escorted shopping expedition six weeks after his admission he suddenly became restless, forgetful, concerned with his own sexual orientation, and preoccupied with the sexual abuse he had suffered as an adolescent. He spent the night pacing around the hostel and was returned to hospital the following day in a highly agitated state. As an emergency measure he was started on chlorpromazine (600 mg per day) but this led to a deterioration in his condition. The same day, while playing pool, he was found to be unable to distinguish right from left, remember the position of the balls, or recognise by sight his fellow patients. He was disorientated in time and place and had clouding of consciousness. He recovered fully within ten minutes.

Neuropsychological testing revealed a full-scale WAIS IQ of 75 (without significant verbal-performance discrepancy), and on other tests (Wisconsin Card Sort test, Rey–Osterrieth test), visiospatial deficits suggestive of an abnormality in his right temporal lobe were found. All medication was stopped to allow for further clinical observation, electroencephalography (EEG) and computerised tomography (CT).

A week after the episode over the pool game, the EEG studies – which included the use of sphenoidal and zygomatic leads – demonstrated occasional abnormal sharp-wave activity in the right anterior temporal horn region and therefore provided no unequivocal evidence of epilepsy. The same day, a standard CT scan (Fig. 1) showed localised ventricular dilation of the right temporal horn, and a specially angled CT scan (Fig. 2) was suggestive of sclerosis in Ammon's horn and cortical atrophy. Two weeks after the EEG studies and CT scans, the patient reported to the nurses that he was 'not himself'. He became tearful and said that he had seen a ghost the previous night. He was convinced his bed was moving, all his teeth were about to fall out, and that he had been transformed into a vampire. He was noted to be disorientated, the left side of his face was drooping, the tongue deviated to the left, and his left arm and leg were partially flaccidly paralysed. His blood pressure was 210/140 mmHg and pulse 50 beats/min. An intracerebral bleed was suspected, but discounted two hours later by the neurosurgeon since the patient's condition, apart from the hypertension and bradycardia, had returned to normal. A further period of close observation, suggested as a precaution by the neurosurgeon, was discontinued the following day, as the blood pressure and pulse had gradually returned to his usual level of 110/80 mmHg and 80/min.

Just over a week later, approximately ten weeks after admission, the nurses reported that, although fully orientated, the patient had appeared over the weekend to have been withdrawn, often drowsy, and almost mute, with a brief period of 'bewilderment' which 'miraculously' disappeared when he was administered sodium amytal, only to return a few hours later. It was thought that this might be due to short bursts of ictal activity. However, 24-h EEG monitoring was not feasible on account of the patient's disturbed state. Instead, this possibility was tested by setting up an intravenous infusion of diazepam while the patient was in one of these 'bewildered' states. The patient was given 2 mg of diazepam intravenously over 10 min, after which his drowsiness abruptly resolved and he suddenly became bright, alert, and conversational. He hugged his favourite nurse and appeared relieved. He was started on carbamazepine, which was maintained at a serum level of 13–18 mg/l. Since that time his mental state has remained normal. After six weeks of graded visits to the hostel he was discharged.

At follow-up, 14 months after his discharge, no further episodes suggestive of either an abnormal mental state or ictal activity have been reported.

Discussion

Epilepsy is essentially a clinical diagnosis and, as illustrated by this report, the EEG may remain equivocal. Up to 10% of cases attending specialised centres and receiving the full battery of EEG procedures have negative test results (Hopkins, 1987), and so CT may be useful as a complementary investigation.

With hindsight, the clinical deterioration in the patient's condition which took place in the tenth week of his admission may have been the result of either chlorpromazine or lithium lowering the seizure threshold. However, the remote possibility of lithium-induced seizures (Jefferson *et al*, 1986) is excluded by the lack of any physical signs of toxicity and the peak serum level (0.8 mmol/l) being within the normal range. The episode of left-sided motor impairment may have been a manifestation of Todd's paralysis. The use of the slow intravenous infusion of diazepam which aborted the episode of perplexity and mood disturbance abruptly, together with the CT findings, suggests that the underlying pathological process, in spite of the equivocal EEG findings, was temporal-lobe sclerosis and consequent epileptiform activity.

It is not known whether there is one focus of epileptiform activity primarily in Ammon's horn, which extends through its abundant motor connections to the neocortex and so produces the Jacksonian-type picture (Rosene & Van Hoesen, 1977; Brodal, 1981), or two foci, the other being somewhere in the post-central cortex.

Hypomanic and manic episodes are rarely reported in epileptic patients (Wolf, 1982); furthermore most reports tend to concentrate on either the interictal (Perez & Trimble, 1980) or the less common post-ictal (Logsdail & Toone, 1988) psychoses. To our knowledge, there are no reports in the literature of cases where episodes of mania have been antecedent to and distinct from the ictal phase of the epileptiform disturbance. In addition, when the manic attacks abated, the epileptiform seizures commenced, and at no time did the two states occur concurrently. Carbamazepine is the drug of first choice in partial (simple and complex) seizures (Hopkins, 1987), and it is increasingly used in the management of both depressive (Post *et al*, 1986) and lithium-resistant manic disorders (Post, 1982; Elphick, 1988). Although other anticonvulsants such as valproate have been shown to have antimanic properties (Emrich *et al*, 1984), no type of seizure activity or abnormal discharge leading to manic symptoms has ever been confirmed by EEG studies. Bearing in mind the absence of a family history of mania in our case, we suggest that the link between epilepsy and mania is more than coincidental. That is, in this case they may share discrete but related biochemical and/or anatomical substrates by way of abnormality of the right temporal lobe which can be improved by anticonvulsant drugs.

Acknowledgements

We thank Dr J. M. Dawson for reporting the CT scan and Professor W. A. Lishman for commenting upon an early draft of this paper.

References

Brodal, A. (1981) *Neurological Anatomy in Relation to Clinical Medicine* (3rd edn), pp. 673–698. Oxford: Oxford University Press.

Elphick, M. (1988) The clinical uses and pharmacology of carbamazepine in psychiatry. *International Clinical Psychopharmacology*, 3, 185–203.

Emrich, H. M., Dose, M. & Von Zerssen, D (1984) Action of sodium valproate and oxcarbazepine in patients with affective disorders. In *Anti-convulsants in Affective Disorders* (eds H M. Emrich, T. Okuma & A. A. Muller), pp. 139–147. Amsterdam: Excerpta Medica

Falret, J. P. (1854) Memoire sur la folie circulaire. *Bulletin de L'Academie Nationale Médicine*, **18**, 382–400.

Flor-Henry, P. (1969) Psychosis and temporal lobe epilepsy. *Epilepsia*, **10**, 363–395.

Hopkins, A. (1987) *Epilepsy* (1st edn). London: Chapman & Hall.

Jefferson, J. W., Greist, J. H. & Ackerman, D L. (1986) *Lithium Encyclopedia for Clinical Practice* (2nd edn). Washington, DC: APA.

Loosdail, S. J. & Toone, B. K. (1988) Post-ictal psychoses. A clinical and phenomenological description. *British Journal of Psychiatry*, **152**, 246–252.

Perez, M. M. & Trimble, M. R. (1980) Epileptic psychosis: diagnostic comparison with process schizophrenia. *British Journal of Psychiatry*,**137**, 245–249.

Post, R. M. (1982) Use of the anti-convulsant carbamazepine in primary and secondary affective illness. *Psychological Medicine*, **12**, 701–704.

——, Uhde, T. W., Roy-Byrne, P. P., *et al* (1986) Antidepressant effects of carbamazepine. *American Journal of Psychiatry*, **143**, 29–34.

Rosene, D. L. & Van Hoesen, G. W. (1977) Hippocampal efferents reach widespread areas of cerebral cortex and amygdala in rhesus monkeys. *Science*, **198**, 315–317.

SLATER, E. & BEARD, A. W. (1963) The schizophrenia-like psychoses of epilepsy. *British Journal of Psychiatry*, 109, 95–150.

WOLF, P. (1982) Manic episodes in epilepsy. In *Advances in Epileptology, XIIIth Epilepsy International Symposium* (eds H. Akimoto, H. Kazamatsuri, M. Seino, *et al*), pp. 237–240. New York: Raven Press.

*Bankole A. Johnson, MB, ChB, MRCPsych, *Honorary Senior Registrar in Psychiatry, Maudsley Hospital, Denmark Hill, London SE5 8AZ*; Lachlan B. Campbell, MB, BS, BMedSc, MRCPsych, *Consultant in Psychiatry, Maudsley Hospital, Denmark Hill, London SE5 8AZ*

*Correspondence

British Journal of Psychiatry (1990), 157, 444–446

Suicidal Behaviour in Severely Mentally Handicapped Patients

RUTH M. WALTERS

Four patients with IQs below 50 displayed suicidal behaviour. This refutes the notion that the severely mentally handicapped are unable to form such an intention.

Suicides of in-patients of psychiatric hospitals are comparatively rare. Goh *et al* (1989) reported estimated figures of 1.3–2.5 per 1000 discharges, suggesting that for any individual psychiatrist experience may be limited.

A national postal inquiry in 1986 by the author to consultant psychiatrists working in mental handicap, into their experience of suicidal behaviour in severely mentally handicapped patients (IQ below 50) elicited 111 replies. Only five psychiatrists recalled patients who had committed suicide, all of whom were described as being mildly mentally handicapped or of borderline ability. All had IQs over 60 and clear diagnoses of psychiatric illness superimposed on their mental handicap. Thirteen recalled patients who had made suicidal attempts or gestures or who had threatened suicide: some were adolescents, one had an IQ of 53, and three may have been of more limited ability. A few respondents thought that severely mentally handicapped people with IQs below 50 would be unable to conceptualise and form an intention to commit suicide, but some considered that the severely self-injurious behaviour of some profoundly handicapped patients with autism could be suicidal. In these cases the behaviour was so forcible and unpredictable that serious injuries resulted. Examples were given of patients with IQs below 25 who repeatedly banged their heads against brick walls and of a young man who threw himself head first through reinforced glass windows.

During nearly 20 years as a consultant at Hanham Hall Hospital (approximately 200 in-patients, mostly male) four male mentally handicapped patients whose IQs were below 50 have exhibited behaviour judged to indicate an intention to endanger their lives.

Case reports

Case 1

WB was admitted to hospital at the age of 16. The cause of his mental handicap is unknown, but he does suffer from epilepsy.

At age 34 his IQ was 46; by 53 years his IQ was 34 and he was judged to have a mental age of five years and ten months.

He was subject to detention under Section 26 of the Mental Health Act 1959, suffering from manic–depressive psychosis between the ages of 46 and 50 years. There were episodes of mild hypomania in the early years of his illness and his episodes of severe depression were accompanied by hypochondriacal and nihilistic delusions with aggression. On several occasions when depressed he absconded from the ward in very inclement weather dressed in pyjamas in order to hide and die. Each time he talked of wanting to be "put in his coffin box". He received five courses of electroconvulsive therapy between the ages of 44 and 54 years but continued to be depressed and delusional with little response to treatment until his death in 1986 at the age of 64.

Case 2

PD suffers from familial mental handicap and epilepsy. He was admitted to hospital under Section 26 of the Mental

Health Act 1959 at the age of 28 in 1973 because of behavioural difficulties and alcohol abuse.

At the age of 24 his IQ was 46; at the age of 28 his IQ was 38 and his mental age six and a half. Three weeks after his admission he was found by nursing staff with a belt tightly fastened around his neck. On questioning he said he had done it "to kill meself, 'cos I can't go home with me mam". On inquiry it was found that he had previously been prevented by other patients from jumping over the stair bannisters. He was uncooperative and was noted to be disruptive, teasing and provoking other patients. His mood was depressed and a diagnosis of acute emotional distress was made.

His mother discharged him, giving 72 hours' notice. He has remained under out-patient care and his behaviour continues to be disturbed. He has been before the court on charges of indecent exposure on three occasions, and twice for causing wilful damage. He is at odds with his neighbours and has himself been assaulted. He has been frequently drunk and wanders around the countryside at night. He is socially untrained and has an inadequate personality. He responds to the occupational and social training available at his adult-training centre but his attendance is erratic.

Case 3

LC has Down's syndrome and epilepsy, and was admitted to hospital aged 21.

At the age of 30 his IQ was 30 and his mental age five years. His speech is limited to phrases and simple sentences, with very poor articulation and a severe stammer, so that no clear history of psychiatric symptoms can be obtained.

His mental state became disturbed at age 18, with screaming outbursts and violent behaviour. At age 21 he had daily episodes when he would scream, become agitated and hyperactive, breaking windows and throwing crockery and furniture. He was often unpredictably violent to staff and patients, causing injuries. Although his mental state improved with treatment in hospital, there were relapses at age 28 and at age 30 lasting several months, necessitating seclusion and cessation of activities. During each episode suicidal behaviour was recorded. In 1975, at age 28, he was found with a knife in his hand and he threatened to take his own life; at the age of 30 he was observed to put his leather belt around his neck and tighten it.

Observed behaviour thought to indicate delusional and hallucinatory experiences includes episodes of hysterical giggling and of talking incoherently to himself, urinating in inappropriate places, tearing clothing, head banging, smelling objects, and hostility to others. It is felt that he probably suffers from a psychotic illness. At age 33 he developed syncopal attacks as a result of heart block and was fitted with a cardiac pacemaker. He remains in hospital and is able to attend occupational therapy classes daily and participate in social activities, his mental state having improved.

Case 4

AH had a mental handicap of unknown aetiology. He was admitted to hospital at ten years of age. His IQ is 38, and he has a mental age of 5 years 11 months.

At age 74 years he was found in the bath with his head tilted backwards trying to submerge. When questioned he said "I'm trying to drown myself". It was noted that he had a urinary-tract infection and had suffered severe recurrent abdominal symptoms from an incisional hernia and distension of the small bowel, long-standing complications of operations for sigmoid volvulus (at age 58) and total colectomy (at age 73). The previous year he had had an operation for cataract extraction and lens implant, and had retired from his work in the hospital sewing room. Staff commented that he seemed to have lost interest in his friends and television in the preceding two or three weeks and was not eating well. He persistently complained about his hernia, his teeth or his spectacles.

At examination he was tearful, withdrawn and monosyllabic, saying he was unhappy and had no friends. A diagnosis of acute depression was made and he responded to treatment with tricyclic antidepressant medication. He died 15 months later of intestinal obstruction and infarction of the ileum.

Discussion

Each of these patients took independent deliberate action when staff were not present. Whether the acts which they committed would have resulted in suicide had they been better able to plan and execute them cannot be known. Their spoken comments at the time seemed to indicate an intention to end their lives. WB and LC were already receiving treatment for their mental illnesses, and AH was diagnosed as suffering from a depressive illness after the event, although his physical illnesses were well recognised and under treatment. These cases demonstrate the risk that even patients with IQs below 50 who are mentally ill can attempt suicide, and are able to appreciate that the outcome of the behaviour could cause death.

All the cases reported fall within categories well recognised and defined as at risk of suicidal behaviour among the general population. Serious self-injurious behaviour in patients who are severely or profoundly retarded or autistic may be potentially self-destructive, but Lovaas (1982) considers that such patients would probably be incapable of an intention to end their lives.

Suicidal behaviour has seldom been studied in the mentally handicapped population, and mental handicap is rarely mentioned in surveys of suicide in hospital populations. Sletten *et al* (1972) recorded three with a final diagnosis of mental retardation in a study of 97 patients who committed suicide in Missouri State hospitals. Carter & Jancar (1984) recorded one patient with affective psychosis for whom a verdict of suicide was returned in their study of sudden deaths. Kaminer *et al* (1987) reported three mentally retarded male adolescents who exhibited suicidal behaviour. There was superimposed psychiatric disorder in all three and two had IQs below 50.

Mentally handicapped people are much more likely to suffer mental illness than the general population, and the more severe the mental handicap the more difficult diagnosis becomes. Many patients cannot complain of psychological symptoms and the illness often presents with a change in behaviour, but sometimes biological symptoms predominate, such as loss of appetite or disturbed sleep. The psychopathology of mental illness in the mentally handicapped may not differ from that in the normal population but will be very difficult to study in patients whose ability to communicate verbally may be severely restricted, even when well. The behaviour of a disinhibited excitable brain-damaged patient resembles that of early hypomania and the psychomotor retardation of a depressive illness may be unrecognised in a patient whose habitual mental processes are slow and undifferentiated. Such patients often do not recognise symptoms of illness in themselves and may not complain spontaneously of symptoms which accompany even common intercurrent physical illnesses, and so diagnosis may be delayed. Behavioural disturbances are also a common presentation of physical illness in this group of patients and this may lead to a misdiagnosis of psychological symptoms. The recognition of symptoms of mental illness in severely mentally handicapped patients depends upon the understanding, the knowledge, and the skill of their carers, nurses and doctors.

More specific attention should be given to the recognition and treatment of psychiatric and physical illness in severely mentally handicapped people in any training courses for nurses and other residential care workers. The risk of suicidal behaviour is small but may present serious management problems, and adequate facilities for appropriate hospital treatment with suitably trained nursing staff for these severely mentally handicapped patients with mental illness or severely self-destructive behaviour will be needed in the future.

Acknowledgement

I thank my psychiatric colleagues who responded with so much interest to my inquiry, particularly those who shared details of their cases. I also thank Dr D. N. Wilson for follow-up information on case 2, and Mrs Eileen Owens for secretarial help.

References

Carter, G. & Jancar, J. (1984) Sudden deaths in the mentally handicapped. *Psychological Medicine*, **14**, 691–695.

Goh, S. E., Salmons, P. H. & Whittington, R. M. (1989) Hospital suicides, are there preventable factors? Profile of the psychiatric hospital suicide. *British Journal of Psychiatry*, **154**, 247–249.

Kaminer, Y., Feinstein, C. & Barrett, R. P. (1987) Suicidal behaviour in mentally retarded adolescents: an overlooked problem. *Child Psychiatry and Human Development*, **18**, 90–94.

Lovaas, O. I. (1982) Comments on self-destructive behaviours. *Analysis and Intervention in Developmental Disabilities*, **2**, 115–124.

Sletten, I. W., Brown, M. L., Evenson, C., *et al* (1972) Suicide in mental hospital patients. *Diseases of the Nervous System*, **33**, 328–334.

Ruth M. Walters, MB, ChB, FRCPsych, DPM, DCH, *former Consultant Psychiatrist at Stoke Park Hospital, 10 Downs Road, Westbury-on-Trym, Bristol BS9 3TX*

British Journal of Psychiatry (1990), **157**, 447–460

Correspondence

Editor: Ian Pullen

Contents: Chronic fatigue syndrome/Life events and the onset of mania/Racism, diagnosis and treatment/Unmet needs for medical care/Compliance with antidepressant medication/Insight/Frontal metabolic deficits in Korsakoff syndrome/Skeleton services/'Vital exhaustion' and myocardial infarction/'Slowly progressive schizophrenia'/Temporal lobe atrophy versus open operculum in Asberger's syndrome/Bipolar affective disorder and anoxic brain damage/Hyperinnervation of orbital frontal cortex in schizophrenia/The prescribing debate (continued).

Chronic fatigue syndrome

SIR: Perhaps the most welcome feature of the paper by Hickie *et al* (*Journal*, April 1990, **156**, 534–540) is the demonstration that physicians and psychiatrists can work profitably together in studies of the still controversial chronic fatigue syndrome (CFS). In addition, the use of standardised instruments and the detailed information given on the sample represent a significant advance on much previous work (David *et al*, 1988). The authors correctly note that the selected nature of the sample limits the generalisations that can be drawn from the study, especially since only a quarter of the 200 eligible patients were actually psychiatrically assessed. However, they still make several conclusions that may not be justified.

The authors suggest that their study contradicts other findings that psychiatric diagnoses are an important feature in patients fulfilling criteria for CFS. Of their sample, 46% fulfilled criteria for major depression during the course of their illness. The figure for current depression was lower, but as 15 of the sample had already completed a treatment trial, and given the remarkable placebo response rate that others have observed in this condition (Gantz & Holmes, 1989), current status is misleading. This figure of 46% is in fact in keeping with other studies of psychopathology and CFS. We studied consecutive referrals to a neurological hospital with severe fatigue that had not been explained despite intensive medical investigation (Wessely & Powell, 1989). In terms of symptoms, these resembled the subjects reported by Dr Hickie *et al*, as nearly all complained of myalgia, paraesthesiae, memory impairment, poor concentration, etc, and 72% reported that their illness had commenced with a viral infection. A total of 47% fulfilled criteria for major depression even when fatigue was excluded as diagnostic symptom (Wessely & Powell, 1989). An identical figure was provided by Manu *et al* (1988), as well as by the two US studies cited by the authors. Only one recent study is discrepant. Millon *et al* (1989) studied CFS patients in a US allergy clinic, who might be thought comparable with those in the current paper. They did not give diagnoses, but found considerably higher scores on the Hamilton Rating Scale for Depression (with 19/24 exceeding the conventional cut-off) than those reported by Dr Hickie *et al*. Thus the findings of Dr Hickie *et al* on rates of depression are in keeping with previous studies, and confirm the importance of routinely assessing mood in chronically fatigued patients.

The other four diagnostic studies also found that an additional (approximately) 20% of subjects fulfilled criteria for other psychiatric disorders, principally anxiety and somatisation disorders. It is not the rates of depression then but the absence of other disorders that is surprising in the study of Dr Hickie *et al*. In our study, the proportion of CFS patients with *any* psychiatric disorder was 72% in total, compared with 36% of controls with fatigue due to neuromuscular disease, matched for length of illness (Wessely & Powell, 1989). That this excess of psychiatric illness in CFS may be a result of the central production of cytokines, as suggested by Dr Hickie *et al*, remains plausible but unproven.

The authors use their findings of low rates of premorbid psychiatric disorder (which does contrast with the American studies cited) to suggest that this is not a risk factor for the development of CFS. However, the design of both this and the American studies does not permit either conclusion. The patients reached the investigators by a complex series of filters, and it is reasonable to assume that general practitioners were more likely to refer patients with severe fatigue and a known psychiatric history elsewhere, reserving an immunological assessment for those with new-onset syndromes. The different health care system in the USA, in which patients have more direct access to specialists, gives a different bias, and may have accounted for the contradictory results. It is difficult to establish the true role of past psychiatric history in the genesis of CFS using hospital-based case-control studies.

The authors do not emphasise somatisation as a significant process in CFS, partly on the results of the Illness Behaviour Questionnaire (IBQ). Given the nature of the sample, it is perhaps unwise to put much credence on the results of a questionnaire that includes such unsubtle questions as "If a disease is brought to your attention do you worry about getting it yourself?". However, the authors make one further important clinical observation. They report that the patients firmly believed in the physical nature of their condition, and rejected any psychological contribution. Such observations are in keeping with other studies of the condition (Imboden *et al*, 1959; Wesseley, 1990), emphasised by the classic quotation on neurasthenia at the start of their paper. This suggests an additional characteristic of many chronic sufferers that may be more clinically important than the presence or absence of either immunological or psychiatric disorder.

Simon Wessely

Institute of Psychiatry
De Crespigny Park
Camberwell
London SE5 8AF

References

David, A., Wessely, S. & Pelosi, A. (1988) Post-viral fatigue: time for a new approach. *British Medical Journal*, **296**, 696–699.

Gantz, N. & Holmes, G. (1989) Treatment of patients with chronic fatigue syndrome. *Drugs*, **38**, 855–862.

Imboden, J , Canter, A. & Cluff, L. (1959). Brucellosis III. Psychologic aspects of delayed convalescence. *Archives of Internal Medicine*, **103**, 406–414.

Manu, P., Matthews, D. & Lane, T. (1988) The mental health of patients with a chief complaint of chronic fatigue: a prospective evaluation and follow-up. *Archives of Internal Medicine*, **148**, 2213–2217.

Millon, C., Salvato, F., Blaney, N., *et al*. (1989) A psychological assessment of chronic fatigue syndrome/chronic Epstein Barr virus patients. *Psychology and Health*, **3**, 131–141.

Wessely, S. & Powell, R. (1989) Fatigue syndromes: a comparison of chronic 'postviral' fatigue with neuromuscular and affective disorders *Journal of Neurology, Neurosurgery and Psychiatry*, **52**, 940–948

Wessely, S. (1990) "Old wine in new bottles": neurasthenia and "ME". *Psychological Medicine*, **20**, 35–53.

Sir: We enjoyed the paper by Hickie *et al* (*Journal*, April 1990, **156**, 534–540), but wish to comment on the interpretation of the results.

Selection of depressed controls should avoid unintentional overlap with CFS patients. Overlap occurs in physical markers such as the VP-1 antigen (a proposed marker of chronic enterovirus infection) with groups such as major depressives (Lynch & Seth, 1989) and those with neuromuscular disorders (Halpin & Wessely, 1989). From further studies we estimate that 30–40% of our depressed controls would show other similar physical abnormalities to CFS patients (Lynch *et al*, 1990, submitted). For these reasons, depressed controls should undergo the same assessment as for CFS and patients with significant physical abnormalities should be excluded.

Secondly, the control group should be homogeneous; in this study, patients possibly with different types and severity of depression are included. We found that in-patient depressed controls had more severe depressive symptoms and fatigue than out-patients, whom CFS patients resemble more in terms of depressive and fatigue severity. We would advocate using out-patients with major depression of milder severity (Lynch & Seth, 1990).

Assessment for both control group and CFS should be initially without medication (antidepressants have quite marked effects on depression and fatigue complaints in previously untreated depressives by the second week of treatment). Other difficulties are whether fatigue should be excluded from diagnostic criteria, as its nature is uncertain in the chronic fatigue syndrome (Wessely & Powell, 1989).

The conclusion that "... there is no evidence that CFS is a variant or expression of a depressive disorder ..." is not justified. The control group used was of typical major depression and findings only hold for this group and not other depressive groups. There are also alternative explanations consistent with the findings on phenomenology and illness behaviour.

Regarding phenomenology; in the analogous situation of atypical facial pain, for example, there is one major symptom of pain, and depressive symptoms may not be obvious. This group would also differ phenomenologically from the depressed controls in Dr Hickie *et al*'s study. This study design cannot in itself refute or confirm whether CFS is an atypical depressive syndrome. The findings on illness behaviour are consistent with those of Wessely & Powell (1989) and Wessely *et al* (1990) in that the differences in attribution of symptoms explain why depressive symptoms such as self-esteem and guilt are more prominent in major depression than CFS. This can be taken to support or refute the above hypothesis concerning CFS. The only certain way of resolving this dilemma is to clarify the nature of fatigue in CFS and its relation to psychological symptoms and/or physical symptoms over time.

SEAN LYNCH

St Charles Hospital
Exmoor Street
London W10 6DZ

RAM SETH

Royal Bethlehem Hospital
Monks Orchard Road
Beckenham, Kent

References

HALPIN, D & WESSELY, S. (1989) VP-1 antigen in chronic postviral fatigue syndrome. *Lancet*, *i*, 1028–1029.

LYNCH, S. P. J. & SETH, R. V. (1989) Postviral fatigue syndrome and the VP-1 antigen. *Lancet*, *ii*, 1160–1161.

—— & —— (1990) Depression and myalgic encephalomyelitis. *Journal of the Royal Society of Medicine* (in press).

WESSELY, S & POWELL, R (1989) Fatigue syndromes a comparison of chronic 'postviral' fatigue with neuromuscular and affective disorders *Journal of Neurology, Neurosurgery and Psychiatry*, **52**, 940–948

——, DOLAN, R & POWELL, R (1990) Attributions and self esteem in depression and the chronic fatigue syndrome. (abstract) Spring Quarterly Meeting, Royal College of Psychiatrists, University of Galway, April 1990

SIR: In response to Dr Wessely and Drs Lynch & Seth, we would like to make the following comments. Firstly, when comparing patients with CFS with those with primary depressive disorders, cases of each disorder should be assigned strictly on the basis of published criteria and should not then be reallocated, post hoc, following the identification of some other potentially important biological parameter. Secondly, we agree that depressive controls should be homogeneous, and that they should have a depressive subtype potentially comparable with CFS. As highlighted by Dr Wessely, other researchers have noted the number of patients with CFS who have sufficient depressive symptoms to reach the criteria for 'major depression'. Appropriately, therefore, we contrasted subjects with major (non-melancholic, non-psychotic) depression with patients with CFS to test the hypothesis that the latter have unrecognised 'major depression'. We demonstrated that patients with CFS differed on key clinical variables (i.e. prevalence of pre-morbid psychiatric disorder, current depression severity and neuroticism). Further, others have demonstrated that patients with CFS do not show proposed biological markers of 'major depression' such as non-suppression on the dexamethasone suppression test (Taerk *et al*, 1987) or shortened latency of rapid-eye-movement sleep (Moldofsky, 1990). Having shown that the hypothesis of CFS being a form of 'major depression' is unlikely, some researchers now propose that such patients have 'atypical' depression, whereby they misattribute their somatic symptoms to physical rather than psychological causes and thereby avoid any personal guilt or fall in self-esteem. However, it seems more likely that CFS is not a primary depressive disorder at all, but rather an acquired neuropsychiatric condition in which depressive and other neurocognitive symptoms are prominent (Lloyd *et al*, 1988, 1990).

Dr Wessely takes issue with the selective nature of our sample. We do not see this as a deficit of the study but rather as a major strength. Surely the key research and clinical issue is to distinguish those patients with CFS from the mass of those with non-specific fatigue and other related states encountered in general medical practice, given that the latter may be inappropriately labelled, by the patients themselves or their doctors, as sufferers of CFS.

The essential psychiatric finding in our report was the low rate of *pre-morbid psychiatric disorder*. Importantly, Goldenberg *et al* (1990) have also reported pre-morbid psychiatric disorder to be infrequent. Dr Wessely is particularly concerned about our low rates of other psychiatric disorders, particularly anxiety and somatisation. It may well be that our strict selection process and the tertiary referral nature of the Immunology and Infectious Disease practices from which the patients with CFS were drawn meant that individuals who did not meet our operational criteria for CFS but had primary psychiatric disorders were treated appropriately elsewhere. Thus, the rate of pre-morbid psychiatric disturbance is likely to be influenced strongly by referral biases.

Dr Wessely raises the difficult issue of 'somatisation', a topic that requires clarification. Those with primary depressive disorders often have multiple somatic complaints, but are generally recognised by their psychiatrists as suffering from depression. As discussed, this is clearly not the case with patients with CFS. The proponents of 'somatisation' argue simply that patients with CFS have a type of communication deviance in which they express their dysphoria primarily in a somatic form. This is at best a highly speculative hypothesis. Surprisingly, Dr Wessely takes issue with our use of a well standardised instrument, the Illness Behaviour Questionnaire (IBQ) to evaluate this concept. Lipowsky (1989) has addressed the difficult issue of somatisation within CFS and has warned psychiatrists to avoid simplistic causal hypotheses. Dr Wessely's final statement that the patient's belief in the physical nature of their condition is clinically more important than either concurrent immunological or psychiatric disorder must surely represent his own point of view and one which, like all others, requires empirical evaluation. Whenever patients with non-specific physical complaints fail to demonstrate evidence of a known disorder, psychiatric evaluation is likely to follow. Often this is appropriate. As has been demonstrated in patients with myasthenia gravis (Nicholson *et al*, 1986), however, such patients are often mislabelled by psychiatrists as 'somatisers' until the actual nature of the disorder is later revealed. To avoid that pitfall, psychiatric diagnoses should be restricted to the identification of 'typical' disorders in patients with positive features of psychological disturbance. More doubtful notions such as 'somatisation' should not be invoked haphazardly to conceal a lack of basic knowledge.

IAN HICKIE
GORDON PARKER

Division of Psychiatry
Prince Henry Hospital
Sydney 2036
Australia

ANDREW LLOYD
DENIS WAKEFIELD

School of Pathology
University of New South Wales
Kensington NSW
Australia

References

GOLDENBERG, D. L., SIMMS, R. W., GEIGER, A., *et al* (1990) High frequency of fibromyalgia in patients with chronic fatigue seen in a primary care practice *Arthritis and Rheumatism*, **33**, 381–387.

LIPOWSKY, Z. J. (1989) Somatisation the concept and its clinical application. *American Journal of Psychiatry*, **145**, 1358–1368

LLOYD, A. R., WAKEFIELD, D., BOUGHTON, C., *et al* (1988) What is myalgic encephalomyelitis? *Lancet*, *i*, 1286–1287.

——, GANDEVIA, S C & HALES, J P. (1990) Muscle performance, voluntary activation, twitch properties and perceived effort in normal subjects and patients with chronic fatigue syndrome. *Brain* (in press).

MOLDOFSKY, H (1990) *CFS and Fibromyalgia First International Conference*. February 1990, Los Angeles, USA.

NICHOLSON, G. A., WILBY, J. & TENNANT, C (1986) Myasthenia gravis: the problem of a "psychiatric" misdiagnosis. *Medical Journal of Australia*, **144**, 632–638.

TAERK, G. S., TONER, B B, SALIT, I. E., *et al* (1987) Depression in patients with neuromyasthenia *International Journal of Psychiatry in Medicine*, **17**, 49–56

Life events and the onset of mania

SIR: The paper by Sclare & Creed (*Journal*, April 1990, **156**, 508–514) makes some extraordinary claims in terms of its methodological virtues over earlier studies, and particularly selects our early work for comparisons. This would, of course, be quite legitimate, but for the fact that they almost systematically misrepresent our work. I quote from my paper: "The checklist was shortened from earlier versions by the omission of events most subjective in quality". In fact, all events referring to deterioration in relationships were omitted; yet Drs Sclare & Creed state: "whereas Ambelas examined events only during the 28 days before admission (interpersonal conflicts, difficulties at work). It is likely that the events collected were the result of the illness rather than contributing to its cause". Had they really read the paper they could not have failed to notice that most of our events were deaths, examinations, births, and physical illness, and only 25% could possibly be related to manic illness. To avoid misclassification of events as independent, whenever even a faint chance of this occurring existed, patients and, of course, all controls were interviewed; yet Drs Sclare & Creed insist on describing our work as a case-note study. They consider our four-week pre-event period insufficient. Logically, however, the further away one moves from the onset, the more over-inclusive the observations become in terms of existence of events, and the weaker the argument for temporal connections. While the longer period is almost unavoidable for studies of depression, where onset tends to be more insidious, this is hardly the case with the loud onset of mania. They also pointed out that admissions in their cohort did not follow onset very quickly; there was a lag of 22 days. In our series, admissions had taken place in most cases within less than a week. This is certainly the result of the fact that their cohort consisted of well established manic–depressive patients, many already on lithium and presumably in environments well used to their problems, while our patients were in their first ever episode when the impact is very different indeed.

This selection of patients was unfortunate in other ways since it was already known by Stern (1944), and was confirmed by our study, that the longer the duration of the illness, the less likely for life events to be associated with further episodes. Drs Sclare & Creed devoted a whole table and paragraph to the argument that patients in their cohort with less than five years of illness were somehow comparable to our first-admission cases. They are not of course; anything after the first episode is qualitatively different. In our study, patients who had more than one episode were handled as old patients in the follow-up. Furthermore, the selection of a sample of quite late manic repeat episodes cannot be used to argue any points about the onset of mania, which is their chosen title; it only tells us about events and their relationship to onset of late manic episodes. With such selection of index cases their results would have been predicted by most of the existing literature, and therefore they do not counter the arguments of previous research. Finally, using patients as their own self-controls is not really the best method of controlling. With such methodological differences, the only paper with which theirs is comparable is that of Kennedy *et al* (1983). The only remaining argument to support their claims of superior methodology is that a good instrument (the LEDS) was used. Unfortunately, good tools are never a substitute for correct selection, critical judgment, and a fair design which risks erring on the side of caution rather than drive to a pre-selected outcome.

However, the publication in its current form raises an even more important issue. The paper was presented at a College meeting, and from the floor I had a chance to point out to the presenter how he was consistently misrepresenting my work and what I considered the faults in sampling to be. It obviously made no difference. It is a sad state of affairs when publishability is allowed to over-ride what one might call etiquette if nothing else, and it becomes even sadder when our academic teachers undersign the deed.

A. Ambelas

Child and Family Psychiatric Services
Westcotes House
Westcotes Drive
Leicester LE3 0QU

References

Ambelas, A. (1987) Life events and mania a special relationship? *British Journal of Psychiatry*, **150**, 235–240.

Kennedy, S., Thompson, R., Stancer, H., *et al* (1983) Life events precipitating mania *British Journal of Psychiatry*, **142**, 358–403.

Stern, E. S. (1944) The psychopathology of manic–depressive disorder and Melancholia. *British Journal of Medical Psychology*, **20**, 20–32

Sir: Our study was essentially a methodological one. Only one previous paper (Chung *et al* 1986) had used the LEDS to collect events and had carefully dated the onset of illness. These authors also failed to find an increased rate of severe events before the onset of mania.

Notwithstanding the above information, Ambelas' study (*Journal*, February 1987, **150**, 235–240) remains a case-note study. He relied on patients' hospital records to elicit life events and to date the onset of mania. We have previously pointed out the drawbacks in this method (*Journal*, June 1987, **150**, 875).

From past studies we had expected to find a relationship between life events and the onset of mania. However, our results indicated that illness episodes in a group of patients with established bipolar disorder did not appear to be preceded by events. Further research using the LEDS in first manic episodes is indicated.

Paul Sclare
Francis Creed

Mental Health Services Unit
Royal Cornhill Hospital
Cornhill Road
Aberdeen AB9 2ZH

Reference

Chung, R. K., Langeluddecke, P. & Tennant, C. (1986) Threatening life events in the onset of schizophrenia, schizophreniform psychosis and hypomania *British Journal of Psychiatry*, **148**, 680–85

Racism, diagnosis and treatment

Sir: In their not unuseful analysis of black and white patients admitted to hospital under Section 136 (*Journal*, March 1990, **156**, 373–378), Dunn & Fahy make the surprising statement that it "challenges the findings of Littlewood & Lipsedge (1981), who suggest that treatment is independent of diagnosis in this [black] group". If they are talking about the paper I think they are (for they have got the title wrong in their references), I should point out that not only did Lipsedge and I not make the statement they claim but the study itself is not concerned with psychiatric treatment at all. More importantly, they are somewhat cavalier about dismissing the possibility of racism in their own data. Selective discrimination on the grounds of perceived ethnic status, whether in medicine or in other institutions, or in everyday encounters, is not something best seen as located in individual attitudes, but in the whole way social action in a society may operate. In the review which they cite (Littlewood, 1986), I was at pains to point out that racial bias in psychiatry is hardly likely to be a conscious and overt penalisation of certain patients. The alternative, however, is not to associate differences in diagnosis and treatment simply with differences located in the patients themselves. We need, painstakingly, to reconstruct how certain assumptions, behaviours and actions of individuals, both the putative patients and the professionals, interact in certain contexts. Such studies require surveys of theoretical assumptions, attitudes to the other, and perceived dangerousness: studies using video material of actual instances, besides an examination of the political economy of psychiatry itself.

To take an instance. One possibility is that doctors, whether through their training or through sharing certain white middle-class professional values, have a tendency to perceive black patients as 'more psychotic' than analogous white patients, and hence are more likely to diagnose them as schizophrenic. We shall find it difficult to examine this in any sort of simple 'double-blind' study, for an extended video film of doctor–patient encounters cannot keep ethnicity as the single dependent variable.

If there is no ideal approach, we can nevertheless look at certain aspects. Do psychiatrists, for instance, carry around with them some sort of schema in which certain behaviours and experiences in one ethnic group are accorded a greater or lesser 'pathological weighting' than in another? To start examining this, admittedly restricted, hypothesis, I wrote a brief clinical vignette prepared in two versions, identical in all respects except that Afro-Caribbean origin was specified in one version but not in the other. The vignette was piloted and modified to offer a picture of what appeared to a group of colleagues could equally well be rated as one of six diagnoses (manic depressive psychosis, major depressive disorder, neurotic/stress reaction, paranoid psychosis, personality disorder, schizophrenia). One or other version of the vignette was randomly given to 338 health professionals: 103 doctors (61 of them psychiatrists), 32 social workers and psychologists, 39 nurses, 48 medical students before they had studied any psychiatry, and 116 medical students after their psychiatry teaching. They were invited to select one diagnosis alone as the most likely. There were three refusals/spoiled returns. The results suggested at this level no trends or significant effects of race on diagnosis, either taken as the separate categories or when collapsed into broader groups such as 'psychosis' or 'depression'. Neither training nor profession affected selection of diagnosis except that the 'medics' (doctors and medical students) were more likely to diagnose schizophrenia in both ethnic groups (taken together, $P < 0.025$).

We cannot, however, now claim that there is no pre-existing mental set whereby qualified psychiatrists or others preferentially associate one or other pattern of psychopathology with a particular ethnic group. While a different vignette may have demonstrated an association, there are more serious problems. The 'neutral' components of a case history may already have an ethnic bias. My own example included repeated unemployment: as this is a more common experience for the black community in general, it will have a greater diagnostic salience for the whites where psychopathology may be presumed to have a greater effect on determining unemployment. Similar objections can be raised in relation to family constellation and so on. In other words, significant distress may be rated as 'normal' in black patients.

While I am thus not claiming any unique advantage for simulated clinical situations, it does seem important to attempt to use vignettes, both written and video, to examine the perception of patients by psychiatrists in more detail. Certainly the perception of 'dangerousness' is amenable to a similar study, inelegant and partial as it may be. The wider issues of the role of psychiatry as one among a number of social control procedures are not so amenable to such an experimental design and could more usefully be examined through an 'interpretative' approach.

Roland Littlewood

Department of Anthropology
University College London
Gower Street
London WC1E 6BT

References

Littlewood, R. (1986) Ethnic minorities and the Mental Health Act. *Bulletin of the Royal College of Psychiatrists*, **10**, 306–308

—— & Lipsedge, M. (1981) Some social and phenomenological characteristics of psychotic immigrants *Psychological Medicine*, **11**, 289–302.

Sir: We are able to support some of the conclusions made by Lloyd & Moodley (*Journal*, June 1990, **156**, 907). We have also examined differences in neuroleptic dosage given to white patients of Caribbean origin and patients of Asian origin (Milner & Hayes, 1990). Data concerning 210 consecutive admissions to the All Saints Hospital in inner-city Birmingham were recorded. The sample comprised 138 (60%) British, 30 (14%) Asian, 34 (16%) Afro-Caribbean and 8 (4%) other subjects.

Of those admitted under section were 14% (19) of the British group, 33% (10) of the Asian group, and 41% (14) of the Afro-Caribbean group. It was found that 85% (32) of the Afro-Caribbean group, 57% (17) of the Asian group and 33% (60) of the British group were diagnosed as suffering from a psychosis (mainly schizophrenia). When each group was matched for diagnosis, there was no difference in mean oral chlorpromazine equivalents given in the first three days (all neuroleptic medication was included in the calculation). Each patient was rated on the Krawiecka scale (Krawiecka *et al*, 1977) in order to obtain a 'psychotic' rating. Those who were diagnosed as suffering from schizophrenia (and other psychoses) received a higher rating irrespective of ethnic group.

This confirms the suggestion made that black inpatients are more likely to receive antipsychotics because they are more likely to be diagnosed as suffering from a psychotic illness, particularly schizophrenia. The higher rate of sectioned admissions may reflect differential access to services among black groups or presumptions about willingness of black patients to receive treatment.

GABRIELLE MILNER

Department of Psychiatry
University of Birmingham
Birmingham B15 2TH

GUILWYM HAYES

Department of Psychiatry
University of Sheffield

References

MILNER, G & HAYES (1990) Ethnicity and initial treatment of psychotic illness. *British Journal of Social and Clinical Psychiatry* (in press).

KRAWIECKA, M , GOLDBERG, D. & VAUGHN, M. (1977) A standardised psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308

Unmet needs for medical care

SIR: We are pleased that Daly (*Journal*, June 1990, **156**, 909) finds our procedure for measuring unmet need potentially useful. We agree that it would be sensible, following our initial study (*Journal*, December 1989, **155**, 777–781), to apply the procedure cautiously to other populations. As we have pointed out in other papers on the same project, we can draw no conclusions beyond those based on our sample (Brewin *et al*, 1987, 1988; Brugha *et al*, 1988; MacCarthy *et al*, 1989). Thus, for example, the particular value of thyroid and liver-function tests in our study cannot be taken yet as a specific recommendation for their general use in this kind of population. It is difficult to see why Dr Daly disagrees with this position.

The surprising conclusion that unmet need was as high in the social services day centres as in the day hospitals was based on findings set out at the end of the fourth paragraph of the results, a point that Dr Daly appears to have overlooked. So far as physical examination is concerned, we did look for the side-effects of medication, including tongue tremor, and thus were able to comment on the patient's need for dental care.

T. S. BRUGHA

Department of Psychiatry
University of Leicester
Leicester LE2 7LX

B. SMITH
J. K. WING

The Maudsley Hospital
London

References

BREWIN, C R., WING, J K., MANGEN, S P. *et al* (1987) Principles and practice of measuring needs in the long-term mentally ill. the MRC needs for care assessment. *Psychological Medicine*, **17**, 971–981.

——, ——, ——, *et al* (1988) Needs for care among the long-term mentally ill: a report from the Camberwell High Contact Survey. *Psychological Medicine*, **18**, 457–468

BRUGHA, T. S., WING, J. K., BREWIN, C. R., *et al* (1988) The problems of people in long-term psychiatric day care the Camberwell High Contact Survey. *Psychological Medicine*, **18**, 443–456.

MACCARTHY, B., WING, J. K., LESAGE, A., *et al* (1989) Needs for care among the relatives of long-term users of day care. *Psychological Medicine*, **19**, 725–736.

Compliance with antidepressant medication

SIR: Depressed patients may comply poorly with drug treatment; non-compliance may range from 15–44% (Myers & Calvert, 1984; Willcox *et al*, 1965). Compliance with newer antidepressants such as mianserin and lofepramine has been little studied in an everyday National Health Service setting. We therefore examined drug compliance in all patients prescribed tricyclics or mianserin and over a three-month period under the care of one consultant psychiatrist (n = 29: 16 out-patients, four day-patients, nine in-patients). Eight patients each received amitriptyline, imipramine and mianserin, and five received lofepramine. The dose of tricyclic varied from 25 mg to 210 mg daily and of mianserin from 30 mg to 90 mg daily. A blood sample was taken 8–12 hours after the previous dose, and the plasma antidepressant level measured by high-pressure liquid chromatography techniques. The plasma level was categorised as absent, low, therapeutic or high (Montgomery *et al*, 1977; Orsulak & Schildkraut, 1979). Depression and anxiety were assessed at the time of sampling by the Krawiecka scale for chronic psychotic patients, and a note made of current side-effects (e.g. dry mouth, blurring of vision, weight gain).

No antidepressant was detected in the serum of only one patient, a day-patient prescribed imipramine. Thus only 3% of the population were clearly non-compliant with their drug therapy. A further seven patients, all receiving tricyclics, had 'low' plasma levels. Non-compliance may be suspected in these patients but pharmacokinetic factors must also be considered. Inspection of the data did not suggest these eight patients differed from the others with regard to sex, hospital status, dose of antidepressant, duration of treatment, current anxiety or depression, and prescriber.

A previous study which also used the 'no antidepressant detected' category assessed non-compliance at 35% (Voris *et al*, 1983). It is not clear why the rate was much lower in the present study. Side-effects can be an important factor in non-compliance, but only six patients had no such complaints. Other factors contributing to compliance may include psychiatric diagnosis, the number and frequency of drugs prescribed, and relief from symptoms causing premature discontinuation.

David Hall
David H. Wiles
Robin G. McCreadie
Department of Clinical Research
Crichton Royal Hospital
Dumfries DG1 4TG

References

Montgomery, S., McAuley, R & Montgomery, D. B. (1977) Relationship between mianserin plasma levels and antidepressant effect in a double-blind trial comparing a single night-time and divided daily dose regimens *British Journal of Clinical Pharmacology*, **5** (suppl. 1), 71–76.

Myers, E D & Calvert, E. J. (1984) Information, compliance and side effects a study of patients on antidepressant medication. *British Journal of Clinical Pharmacology*, **17**, 21–25.

Orsulak, P J & Schildkraut, J J (1979) Guidelines for therapeutic monitoring of tricyclic antidepressant plasma levels *Therapeutic Drug Monitoring*, **1**, 199–208

Voris, J C, Morin, C. & Kiel, J S (1983) Monitoring outpatients' plasma antidepressant-drug concentrations as a measure of compliance *American Journal of Hospital Pharmacy*, **40**, 119–120.

Willcox, P R C., Gillan, R & Hare, E H (1965) Do psychiatric patients take their drugs? *British Medical Journal*, *ii*, 790–792

Insight

Sir: David (*Journal*, June 1990, **156**, 798–808) discusses three aspects of insight that are commonly recognised in the psychiatric mental state: treatment compliance, awareness of illness, and correct relabelling of psychotic experiences. David appropriately considers each of these to be dimensions, rather than 'all or none'. However, even this approach is an over-simplification. None of these three sorts of insight have been satisfactorily reduced to a quantitative scale, since each involves a number of different qualitative judgements. Furthermore, by carefully examining these judgements, we find that these three different aspects of insight are, in fact, crucially linked.

Consider what is necessary to 'relabel a psychotic experience correctly'. When a patient 'hears voices', he/she is not *imagining* that he hears voices: he is having the perceptual experience of hearing the sound of a voice – he is hearing a 'real' sound. To relabel the experience correctly, he has to recognise that for the experience to be 'normal', there must, in addition, be an identifiable source to the sound, and he has to establish that there is no such source. However, even this is not enough: he must also recognise the correct explanation for his abnormal experience rather than invoking other explanations (of varying degrees of plausibility) to reconcile this experience with his knowledge and beliefs about reality.

To be compliant with treatment, or to admit to an awareness of illness, involves an equally complex chain of decisions concerning, among other things, the patient's attitude to the current practice of psychiatry. However, *in common* to all three aspects of insight is this issue whether the patient recognises that he is, in some way, functioning abnormally.

The interesting thing is that many patients *do* realise that their experiences (or that they themselves) are abnormal in some way – but they may *not* go so far as to 'correctly relabel' their psychotic experience, and so would not score on Dr David's schedule. We know that patients are often aware of this abnormality because it is common to see a patient invoking elaborate explanations for his psychotic phenomena. However, the explanations that are invoked may, themselves, be at odds with reality. Thus 'double awareness' or double orientation (i.e. simultaneously entertaining two beliefs that are irreconcilable, given the currently accepted limits of science) is actually very common, either because the patient accepts his psychotic experience as real, yet not in keeping with reality, or because the patient accepts a bizarre explanation for his experience.

This awareness by the patient of the 'oddness' of his experience is very important, because it probably results both in the patient's awareness that he is ill and (crucially) in the motivation for treatment compliance – whether or not the patient takes the step of 'relabelling' his psychotic experience. I would suggest, therefore, that measuring the patient's degree of insight is of great value in assessment and in therapy, both physical and psychological. However, there are many more detailed questions to be asked than have been covered in Dr David's schedule.

Francis Klemperer
Research Fellow
Guy's Hospital
London SE1 9RT

Frontal metabolic deficits in Korsakoff syndrome

Sir: Lishman's review (*Journal*, May 1990, **156**, 635–644) emphasises importantly the spectrum of brain damage found in alcoholics and, in particular, the accumulating evidence that Korsakoff patients not only have subcortical lesions, but in most cases significant cortical damage also. In the review, Lishman discussed the support for this from structural imaging methods such as X-ray computerised tomography (CT). Unfortunately, CT provides little or no information on regional brain *function*.

Several functioning imaging studies show that regional cerebral blood flow (rCBF), an index of cerebral metabolism, is reduced in patients who have taken excessive quantities of alcohol over long periods (e.g. Berglund, 1981). Positron emission tomography (PET) and single photon emission tomography (SPET) have been used to investigate regional brain metabolism in Korsakoff patients. In the study by Hata *et al* (1987), quoted by Lishman, rCBF in a small group of possible Korsakoff patients returned to normal following abstinence and thiamine. However, other evidence from cohorts of patients carefully defined as having chronic Korsakoff syndrome suggests that there is a permanent impairment of function in the cortex, particularly in the frontal regions, even after prolonged abstinence and thiamine treatment. Non-tomographic cortical measures of rCBF (Hunter *et al* 1989*a*) and SPET using the flow tracer ^{99m}Tc–hexamethyl propyleneamine oxime (^{99m}Tc HMPAO; Hunter *et al*, 1989*b*) indicate significantly impaired perfusion in frontal cortex in abstinent Korsakoff subjects. Furthermore, significant correlations were detected between impaired cognitive performance, particularly memory function, and rCBF measured using SPET in frontal cortex of Korsakoff patients (Hunter *et al*, 1989*b*). These findings in Korsakoff patients are in marked contrast to measures of rCBF using SPET in pre-senile Alzheimer patients, where perfusion deficits were marked in posterior temporal and parietal cortical regions, areas that appeared relatively normal in Korsakoff patients (Hunter *et al*, 1989*b*). These results are consistent with those from PET, where absolute rates of regional cerebral glucose metabolism were reduced in cortical and subcortical regions in Korsakoff patients (Kessler *et al*, 1984).

There are difficulties in the interpretation of functional scans, however. For instance, the finding of frontal reductions in rCBF or glucose metabolism could mean that a normal tissue mass has reduced neuronal activity, or that a reduced tissue mass has normal levels of activity or some mixture of the two. The CT and neuropathological studies quoted by Lishman showing structural loss of grey and white matter in frontal lobes, suggest that reduced rCBF in this region is likely, at least in part, to reflect some contribution from atrophic change. Further studies are required to assess the size of this contribution. Nevertheless, functional imaging techniques, and in particular SPET and PET, provide important evidence of frontal metabolic impairment in Korsakoff syndrome. Such techniques may demonstrate a pathophysiological basis for the well recognised clinical features of Korsakoff syndrome, such as behavioural change, apathy and loss of initiative, which in the past were presumed from lesion studies to be frontal in origin.

ROBERT HUNTER

MRC Brain Metabolism Unit
Royal Edinburgh Hospital
Morningside Park
Edinburgh EH10 5HF

References

BERGLUND, M. (1981) Cerebral blood flow in chronic alcoholics *Alcoholism Clinical and Experimental Research*, **5**, 295–303.

HATA, T, MEYER, J. S., TANAHASHI, N., *et al* (1987) Three-dimensional mapping of local cerebral perfusion in alcoholic encephalopathy with and without Wernicke-Korsakoff syndrome. *Journal of Cerebral Blood Flow and Metabolism*, **7**, 35–44

HUNTER, R, MERRICK, M V., FERRINGTON, C., *et al* (1989*a*) Cerebral vascular transit time in Alzheimer's disease and Korsakoff's psychosis and its relation to cognitive function. *British Journal of Psychiatry*, **154**, 790–796.

——, MCCLUSKIE, R., WYPER, D., *et al* (1989*b*) The pattern of function-related regional cerebral blood flow investigated by single photon emission tomography with ^{99m}Tc-HMPAO in patients with presenile Alzheimer's disease and Korsakoff's psychosis. *Psychological Medicine*, **19**, 847–855

KESSLER, R M, PARKER, E S., CLARK, C S, *et al* (1984) Regional cerebral glucose metabolism in patients with alcoholic Korsakoff syndrome. *Society for Neuroscience*, **10**, 541

Skeleton services

SIR: We undertook a Christmas census at the Walsgrave Psychiatric Unit of all in-patients present at midnight on Christmas Eve, 1989. Twelve of the patients were over the age of 65 years with functional mental illness, six fulfilling DSM–III critera for major depressive disorder, one for alcohol dependence, one for personality disorder, four for psychoses not otherwise classified and one inappropriately admitted because of medical problems. The severity of illness was the reason for staying in hospital over Christmas in nine of the patients, mainly because of poor eating and drinking or suicidal intentions. Three of the patients were in hospital because of social reasons, one because the main carer was too ill to look after the patient, and two because Part-III homes were unable to cope with them because of decreased staffing levels over the Christmas period. In addition to these three patients, two of the nine patients staying because of medical reasons were unmanageable by their families simply because they exhibited a degree of agitation; they would probably have been able to go for a short period of leave had there been suitable community services in operation.

We are rather concerned that five of the patients kept in hospital over the Christmas period were there mainly because of the low levels of staffing of social services and community psychiatric services at Christmas. Although ideally everybody would like time off work to be with their families at this time of year, can the skeleton services provided currently be ethically justified in the light of the figures from our census?

C. G. Ballard
R. N. Chithiramohan
S. Handy

Department of Psychiatry
Walsgrave Hospital
Coventry CV2 2DX

'Vital exhaustion' and myocardial infarction

Sir: Appels (*Journal*, April 1990, **156**, 465–471) draws attention to the mental precursors of myocardial infarction. We would like to add the findings of our recent study of 50 Indian patients with acute myocardial infarction and 50 age- and sex-matched healthy controls. The frequency and intensity of type-A behaviour, life events occurring during the period of one year preceding myocardial infarction and the levels of anxiety and depression following myocardial infarction were studied.

According to Jenkins activity survey (form C; Jenkins *et al*, 1979), type-A behaviour was detected in 72% of patients as compared with 18% of the control group. With regard to different components of type-A behaviour, type A and factor S (measuring speed and impatience) were significantly higher in the study group, while factors J (measuring job involvement) and H (measuring hard driving and competitiveness) did not differ significantly between the two groups. The patients also had significantly greater mean frequency of stress scores on the Presumptive Stressful Life Events Scale (Singh *et al*, 1983) which measures both total life events and subgroups of this – the desirable, undesirable, personal, impersonal and ambiguous life events.

The stressful life events which discriminated the patients from controls were (in decreasing significance): marital conflicts, financial loss, change in working condition, major personal illness, trouble at work, death of a family member, and change/expansion of business. The mean scores of anxiety (as assessed by the Hamilton Rating Scale) and depression (as measured on Beck's Depression Inventory) showed statistically significant falls over the periods of two weeks, one month and three months after infarction, but became insignificant subsequently.

In conclusion, type-A behaviour and stressful life events are significantly associated with the risk of myocardial infarction.

M. S. Bhatia

Department of Psychiatry
University College of Medical Sciences
Shahdara, Delhi-110095, India

Ajit Tiwari

Department of Medicine
RML Hospital
New Delhi-110001, India

References

Appels, A (1990) Mental precursors of myocardial infarction *British Journal of Psychiatry*, **156**, 465–471.

Jenkins, C D., Zyzanski, S J. & Rosenman, R H. (1979) *Manual for the Jenkin's Activity Survey Form C*. New York Academic Press

Singh, G., Kaur, D. & Kaur, H. (1983) *Presumptive Stressful Life Events Scale*. Agra National Psychological Corporation

Sir: The 'vital exhaustion' preceding myocardial infarction in men discussed by Appels (*Journal*, April 1990, **156**, 465–471) is supported by a correlation of type B with somatic risk factors of coronary heart disease in adolescent boys (Keltikangas-Jarvinen & Jokinen, 1989). It is also supported by speech hesitation pauses of one second or more occurring more than twice a minute, a component of type-B style but not of global type-A, predicting a six-fold increase in coronary incidence in two groups of normal coronary-prone men followed prospectively for ten years (Case *et al*, 1988). The correlation of the 'traditional' type-A components like impatience and sense of hurry with somatic risk factors of coronary heart disease in adolescent girls (Keltikangas-Jarvinen & Jokinen, 1989) may have been due to their higher metabolic rate (Baxter *et al*, 1988) and dopamine lateralised to the right hemisphere (Friedman; *Journal*, February 1990, **156**, 285). The role of gender-related hemispheric differences is supported by increased dopaminergic activity manifested by mania in a 76-year-old woman with improved Parkinsonism (Menza & Chastka, 1989) compared with reduction of obsessive slowness in a 17-year-old boy in response to fluoxetine, with a return to normality of decreased tracer deposition in the right basal ganglia and adjacent temporal lobe (Hamlin *et al*, 1989).

Ernest H. Friedman

1831 Forest Hills Boulevard
East Cleveland
OH 44112-4313

References

BAXTER, L R., SCHWARTZ, J. M., MAZZIOTTA, J. C, *et al* (1988) Cerebral glucose rates in nondepressed patients with obsessive-compulsive disorder. *American Journal of Psychiatry*, **145**, 1560–1563.

CASE, R B., FRIEDMAN, E. H & FRIEDMAN, M. (1988) Type A behavior (letters and reply) *American Heart Journal*, **116**, 1653–1657.

KELTIKANGAS-JARVINEN, L. & JOKINEN, J. (1989) Type A behavior, coping mechanisms and emotions related to somatic risk factors of coronary heart disease in adolescents. *Journal of Psychosomatic Research*, **33**, 17–27.

HAMLIN, C. L, SWAYZE, L. C & LIEBOWITZ, M R (1989) Striatal IMP-spect decrease in obsessive-compulsive disorder, normalized by pharmacotherapy. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, **2**, 290–300.

MENZA, M A. & CHASTKA, E (1989) Idiopathic Parkinson's disease and mania: the effect of mania on the movement disorder. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, **2**, 301–305.

'Slowly progressive schizophrenia'

SIR: By criticising the concept of slowly progressive or sluggish schizophrenia adopted by Snezhnevsky and his school in the USSR, Shafran *et al* (*Journal*, August 1989, **155**, 174–177) reject not only the existence of this concept but also that of simple schizophrenia. The main support for the authors' idea comes from DSM–III. They point out that "Simple schizophrenia by name has disappeared from DSM–III–R", and that the concept of latent schizophrenia which appeared in DSM–II "was, of course, radically changed for DSM–III".

It is amazing that the authors do not even mention ICD–9 which is the internationally approved classification and which describes simple schizophrenia. In the preliminary version of ICD–10 (World Health Organization, 1987) it is included again under F21.1. Black & Boffeli (1989) suggest that it might even reappear in the next version of DSM. It should not be forgotten that the rejection of simple schizophrenia in DSM was due mainly to the overdiagnosis of this disorder in the USA. Even if a diagnosis can lead to mistakes, it does not mean that it does not exist.

An important argument used by Shafran against simple schizophrenia is Schneider's concept of the first-rank symptoms: "... the Schneiderian backlash put an end to his [Bleulers'] concept of schizophrenia". Although invaluable for the diagnosis and for research purposes, the importance of first-rank symptoms is clearly overestimated. It is worth remembering Schneider's (1950) own words: "... which we call first rank symptoms not because we regard them as 'basic disturbances' but because they have special significance for the *diagnosis* both against nonpsychotic mental abnormalities, as well as against cyclothymia.... Nothing is said about the theory of schizophrenia, unlike Bleuler's basic and accessory symptoms or the primary and secondary symptoms of other authors.... First rank symptoms do not have to be present for the diagnosis of schizophrenia..."

Over-reliance on first-rank symptoms (and on positive symptoms as a whole) could lead to diagnosing only one form – paranoid schizophrenia. The characteristic 'negative' features of residual and simple schizophrenia, which are also prominent in the hebephrenic form, are at least as important in diagnosing and understanding schizophrenic illness. If more attention is paid to the variety of symptoms, some of them very subtle, more diagnostic entities will emerge. Not only Snezhnevsky has described many forms. Leonhard (1986), for instance, listed 19 subforms of schizophrenia apart from the three cycloid psychoses. Such efforts for greater diagnostic precision should not be discouraged, even if they make life more difficult.

Simple schizophrenia has the right to exist because here we see the basic symptoms which are more specific of the schizophrenic deterioration than anything else. They include the peculiar change in the personality which is not seen anywhere else in human pathology, namely that the person loses the core of their personality and is completely different in their reactions to important things in their surroundings. In this respect we could probably cite Ey *et al* (1974): "... schizophrenia is not at the beginning of the evolution but at its end".

GEORGE KIROV

Warlingham Park Hospital
Warlingham
Surrey CR3 9YR

References

BLACK, D W. & BOFFELI, T. J. (1989) Simple schizophrenia past, present and future. *American Journal of Psychiatry*, **146**, 1267–1273

EY, H., BERNARD, P. & BRISSET, C. (1974) *Manuel de Psychiatrie*, p. 564. Paris: Masson

LEONHARD, K. (1986) *Aufteilung der Endogenen Psychosen und ihre differenzierte Atiologie* 6 Auflage Berlin: Academie-Verlag

SCHNEIDER, K (1950) *Klinische Psychopathologie*. 3 Auflage Stuttgart: Georg Thieme Verlag. (p. 138–139)

WORLD HEALTH ORGANIZATION (1987) *ICD–10 1986 Draft of Chapter V*. Geneva: WHO Division of Mental Health.

Temporal lobe atrophy versus open operculum in Asperger's syndrome

SIR: The report by Jones & Kerwin (*Journal*, April 1990, **156**, 570–572) concluded that the patient with Asperger syndrome had atrophy of the left temporal lobe. On the basis of the computerised tomography scan published in their article, we suggest an alternative explanation. The enlarged distance from the temporal tip to the inferior frontal gyrus is consistent with the radiological diagnosis of 'open operculum'.

The diagnosis of open operculum suggests an underlying mechanism distinct from that which results in atrophy. Tatum *et al* (1990) suggest that an open operculum is the result of abnormal neuronal migration during the first part of gestation. They gave case reports of four infants with open opercula. In one infant, examined neuropathologically, they also detected abnormalities in the formation of the cerebral cortical gyri (i.e. macrogyria and micropolygyria). Another infant had Acardi syndrome, known to be associated with micropolygyria and heterotopias. These cerebral cortical anomalies are most likely the result of abnormalities in neuronal migration *in utero* (Larroche, 1984).

We recently detected similar cerebral cortical anomalies consistent with defects in neuronal migration in seven of 13 high functioning autistic males (Piven *et al*, 1990). We performed magnetic resonance imaging (MRI) on 13 autistic men and 13 controls (comparable on age and IQ) and found five autistic individuals with polymicrogyria, one with macrogyria, and one with schizencephaly. In another preliminary study, Berthier *et al* (1990) reported developmental cortical anomalies in two subjects with Asperger's syndrome studied with MRI. One subject was also noted to have a MRI scan consistent with opercular dysplasia.

Thus, the radiological diagnosis of open operculum is consistent with our studies of radiological abnormalities of the brain in autism and Asperger's syndrome. Further, the clinical course which would be expected with a developmental brain abnormality is more consistent with the natural course of both Asperger's syndrome and autism than that which would be expected with the occurrence of atrophy.

JOSEPH PIVEN
SERGIO STARKSTEIN

Department of Psychiatry
Johns Hopkins University School of Medicine
Baltimore, Maryland, USA

MARCELO L. BERTHIER

Institute of Neurological Research
University of Buenos Aires
Buenos Aires, Argentina

References

BERTHIER, M. L., STARKSTEIN, S E. & LEIGUARDA, R. (1990) Developmental anomalies in Asperger's syndrome: neuroradiological findings in two patients. *Journal of Neuropsychiatry*, 2, 197–201

LARROCHE, J. C. (1984) Malformations of the nervous system In *Greenfield's Neuropathology* (eds J. Adams, J. Corsellis & L. Duchen). New York Wiley.

PIVEN, J , BERTHIER, M. L., STARKSTEIN, S. E., *et al* (1990) Magnetic resonance imaging in autism. evidence for a defect of cerebral cortical development *American Journal of Psychiatry*, **147**, 734–739

TATUM, W O , COKER, S B., GHOBRIAL, M , *et al* (1990) The open opercular sign: diagnosis and significance *Annals of Neurology*, **25**, 196–199.

Bipolar affective disorder and anoxic brain damage

SIR: Collins & Jacobson (*Journal,* May 1990, **156**, 736–740) discussed the case of a bipolar patient who developed mild brain damage from anoxia after attempting suicide by hanging. The discussion helped resolve many of the complexities of the diagnosis and treatment of this patient who had concomitant neurological and functional abnormalities. We suggest an additional interpretation of the patient's condition, which may contribute to an understanding of the evolution of her symptoms.

The authors reported that during the two months following the suicide attempt Ms A showed mutism and poor self-care. Although these symptoms can be attributed to depression, they occurred within a constellation of several other neurological abnormalities. Also, the psychiatric staff did not observe frank depressive symptoms. The patient was free from vegetative symptoms of depression. Her sleep and appetite were unremarkable and there was no diurnal variation in her behaviour. It is probable that the patient's depression improved for a period of a few months after her suicide attempt. Although one cannot rule out the effect of the electroconvulsive therapy that the patient was receiving before the suicide attempt, it is possible that the suicide attempt itself had an impact on the patient's mood.

Improvement of mood after a failed suicide attempt is a fairly common but probably temporary event. Lesse (1967) described the positive effects of suicidal behaviour on the mood. These 'apparent remissions' after suicide attempts can be attributed to the cathartic effect of suicide and its role in stimulating an empathic and supportive environment and to the emotional impact of the survival.

We previously reported the observation of manic and hypomanic switches immediately or shortly after a suicide attempt by hanging in four depressed patients. Two of these patients were previously diagnosed as being bipolar (Bourgeois *et al*, 1985). In our review we cited the observation of the 19th-century psychiatrists that melancholics who survived suicide by hanging had their mental state improved These authors were attributing the improvement to asphyxia. Moreover, 'carbonarcose' or anoxic shock (coma induced by letting patients inhale a mixture of 30% carbon monoxide and 70% oxygen) was practiced by several psychiatrists in 1946 as a method of treatment of depression (Bourgeois, 1967). More recently, endogenous depressed patients were found to have a lower response to carbon dioxide (CO_2) inhalation. Damas-Mora *et al* (1978) hypothesised that depressed patients have hyposensitivity to CO_2. However, the relationship between anoxia, CO_2 sensitivity and depression is still speculative. Also, it is reported that during periods of acute ischaemia there is a progressive increase in calcium entry inside the cell, an increase in catecholamine release, and a rapid accumulation of cyclic AMP (Palmer, 1985). These changes may play a role in precipitating a mood change after suicide attempt by hanging.

In summary, Ms A probably had a partial and temporary change of depressive symptoms after her suicide attempt. This increased the complexity of her clinical picture. Whether the effect is biological, psychological or both, such possible effects need always be considered in the evaluation of the mental status of depressed patients who fail in their suicide attempt. Further research needs to be conducted on the effect of failed suicide on the mood as well as on the possible interesting relationship between anoxia and CO_2 in depression.

MICHEL J. CALACHE
Department of Psychiatry
Southern Illinois University School of Medicine
707 N. Rutledge Street
Springfield, Illinois 62794-9230, USA
MARC BOURGEOIS
University of Bordeaux
Centre Carreire
121, rue de la Bechade
Bordeaux, 33076 Cedex, France

References

BOURGEOIS, M (1967) Melancolies hypoxohypercapniques *Annales Medico-Psychologiques*, 125, 422–429

——, DEGEILH, B., CALACHE, M , *et al* (1985) Virage maniaque après suicide violant (pendaison) chez 4 desprimes melancoliques *Annales Medico-Psychologiqes*, 143, 975–985.

DAMAS-MORA, J , JENNER, F A , SNEDDON, J., *et al* (1978) Ventilatory responses to carbon dioxide in syndromes of depression *Journal of Psychosomatic Research*, 23, 473–476.

LESSE, S. (1967) Apparent remissions in depressed suicidal patients. *Journal of Nervous Mental Disorders*, 144, 291–296.

PALMER, G. C. (1985) Cyclic nucleotides in stroke and related cerebrovascular disorders *Life Sciences*, 36, 1995–2006.

Hyperinnervation of orbital frontal cortex in schizophrenia

SIR: Waddington (*Journal*, May 1990, **156**, 615–619) is rightly intrigued by Schetzman *et al*'s (1988) findings of increased resting glucose consumption (hypermetabolism) in the frontal lobe of neuroleptic-naive schizophrenics. This contrasts with several earlier reports of hypofrontality in previously treated schizophrenics. Dr Waddington discusses the speculation that hyperfrontality could indicate hyperinnervation of frontal cortex. He points out that this is a variant of the hypothesis that schizophrenia involves a fault in the normal developmental process of synaptic elimination. However, rather than discussing the relevant neurochemical evidence, Waddington reviews studies of the peripheral neural cell adhesion molecule (NCAM) whose relationship to the impaired synaptic elimination story seems obscure.

We presented direct evidence compatible with an excessive glutamatergic innervation of orbital frontal cortex in schizophrenia (Deakin *et al*, 1989). *In vitro* binding of ^{3}HD-aspartate, a ligand that binds to glutamate uptake sites, and of ^{3}H-kainate, a glutamate receptor ligand, were both significantly increased in orbital frontal cortex from left and right sides of schizophrenic brains obtained at post-mortem. We have since found increased ^{3}H-TCP binding to another type of glutamate receptor which is again localised to orbital frontal cortex.

Independent corroboration of increased frontal cortical ^{3}H-kainate binding is described in a Japanese brain series (Toru *et al*, 1988). Furthermore, G. P. Reynolds (personal communication, 1989) has recently found increased concentrations of glutamate itself, which are localised to orbital frontal cortex in schizophrenic samples.

We pointed out that an increased number of glutamate synapses in orbital frontal cortex could explain increased ligand binding to pre- and post-synaptic sites (Deaken *et al*, 1989). Since the changes were bilateral, we further suggested that an excessive mutual innervation of one orbital frontal cortex by the other could be the origin of the additional synapses. We suggested that the cause was an arrest in the normal developmental process which eliminates immature transient projections. We agree with Dr Waddington that more imaging studies in drug-naive subjects are needed to identify disease-related changes in cerebral function, but direct studies of the neurochemical anatomy of the brain are needed to identify the primary pathology.

J. F. W. DEAKIN
P. SLATER
M. D C. SIMPSON
M. C. ROYSTON
Mental Illness Research Unit
Departments of Psychiatry and Physiological Sciences
University of Manchester, Manchester M13 9PT

References

DEAKIN, J. F W., SLATER, P., SIMPSON, M. D. C., *et al* (1989) Frontal cortical and left temporal glutamate dysfunction in schizophrenia. *Journal of Neurochemistry*, **52**, 1781–1786.

SCHETZMAN, H , NAHMIAS, C , GARNETT, S., *et al* (1988) Effect of neuroleptics on altered cerebral glucose metabolism in schizophrenia. *Archives of General Psychiatry*, **45**, 523–532.

TORU, M., WATANABE, S., SHIBUYA, H., *et al* (1988) Neurotransmitters, receptors and neuropeptides in post-mortem brains of chronic schizophrenic patients. *Acta Psychiatrica Scandinavica*, **78**, 121–137.

The prescribing debate (continued)

SIR: In my previous letter (*Journal*, October 1989, **155**, 566), I had hoped, as requested, to stimulate debate and articles such as Fleming's (1990) in the *Bulletin*. Since Johns (*Journal*, January 1990, **156**, 129) went to the trouble of reading my two references, he may have noted that the first referred to *empirical experiments* (e.g. Stimson *et al*, 1978; Vaillant, 1984). These, replicated more widely and collated by Schneider (1988), show that no external agency expedites the ending of addiction, not even life-events (Sobell, 1990). However, there is evidence that prescribing reduces the harm done to addicts and others and reduces the incidence of addiction, i.e. is preventative (see Marks, 1990).

The second reference was not my "restated well known view", but the balanced and considered opinion of the Editor of the *Lancet*. Dr Johns might also reflect on the fact that there is *no* evidence that *not* prescribing is helpful. Indeed, such evidence as we have (the British System 1920–1960 versus the prohibition 1960–1990) suggests the opposite. I merely suggest experiments: our experiment in Widnes reduced drug-taking and stopped AIDS. None of our injectors, all of whom have been tested, are infected. London's policy of restricting prescribing has seen a rise in HIV infection *and* drugtaking. The onus is on Dr Johns and his colleagues to refute (or replicate!) our findings.

J. A. MARKS

Chapel Street Clinic
Chapel Street
Widnes WA8 7RE

References

FLEMING, P. M. (1990) On Dutch drug policy *Bulletin of Royal College of Psychiatrists*, **14**, 161–162.

MARKS, J. A. (1990) The practice of controlled availability In *Proceedings of the Fifth International Conference on Treatment of Addictive Behaviours (ICTAB-5)* (ed N Heather, W R. Miller & J. G Greeley). Sydney: Pergamon Press.

SCHNEIDER, W (1988) Zur Frage Von Ausstiegschanzen und Selbstheilung Bei Opiatabhangigkeit *Suchtgefahren*, **34**, 472–490

SOBELL, L. C. (1990) The aftermath of heresy drinking and life events. In *Proceedings of the Fifth International Conference on Treatment of Addictive Behaviours (ICTAB-5)* (ed. N Heather, W. R Miller & J. G. Greeley). Sydney: Pergamon Press

STIMSON, G. V , OPPENHEIMER, E., THORLEY, A (1978) Seven year follow-up of heroin addicts. *British Medical Journal*, *i*, 11–90

VAILLANT, G. E. (1984) *The Natural History of Addiction* Dent Memorial Lecture, Centennial Symposium of the Society for the Study of Addiction London: Audio-Stat

CORRIGENDUM

Journal, July 1990, **157**, 25–33 (G. Thornicroft). The last sentence in the penultimate paragraph on page 32 should read "Finally, when ethically acceptable..."

A HUNDRED YEARS AGO

The treatment of alcoholism by strychnine

Dr Pombrak, writing in the Meditsinskoē Obozrēnie on alcoholism, describes seven cases treated by hypodermic injections of strychnine – a method that seems especially in favour in Russia, where, however, it must be remembered that drunkenness presents as a rule in forms somewhat different from those prevalent in this country. Dr Pombrak found strychnine a very valuable remedy, both in cases of chronic alcoholism and in those of dipsomania, not merely curing the attacks, but abolishing the desire for drink. Even attacks of delirium tremens were influenced beneficially. The treatment must be carried out in a systematic manner, and must frequently be kept up for a very considerable period. As to the dose, Dr Pombrak in cases of moderate severity commenced with one-thirtieth of a grain, in more serious ones with one-fifteenth. He found that while the treatment was being carried out there was no necessity to order the patients to abstain from the use of spirits, as they always did so of their own accord.

Reference

Lancet, 3 May 1890, 980.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey

British Journal of Psychiatry (1990), **157**, 461–463

Reading About . . .

Neuropsychology

Clinical neuropsychology is concerned with elucidating the relationship between abnormal cognitive functioning and the neuroanatomical correlates in patients suffering from neurological disease. Its main aim is to contribute to diagnosis of neurological disease, monitoring severity and change in function. It also plays a major role in differentiating between organic and non-organic disorders, is a necessary guide for developing appropriate rehabilitation programmes, and contributes to our understanding of normal cognition. This article aims to highlight those books and papers from selected areas of neuropsychology that are of particular interest.

The increase in interest in neuropsychology is reflected in a number of good general texts in recent years. The major neuropsychological disorders are reviewed in Heilman & Valenstein's 1985 book, which provides methods of assessment, diagnosis and treatment where relevant, and addresses the pathophysiology and neuropsychological mechanisms underlying the disorders. Several chapters in Robbins & Cooper's 1988 book are also of interest, particularly that by McCarthy which gives an excellent overview of the major neuropsychological syndromes from a diagnostic perspective. The methods of assessment are discussed and the anatomical correlates of the particular types of impairment are specified when known. The applications of neuropsychology to clinical conditions that encompass the interactions between neurology and psychiatry can be found in Grant & Adams (1986). In addition, Shallice (1988) provides an interesting and detailed account of cognitive neuropsychology. He considers what can be inferred about the operation of the normal cognitive system including perception, memory, and language from the study of the cognitive impairment arising from neurological damage and disease. The problems of making valid theoretical inferences from impairment to normal function are discussed. His aim is to introduce the methods of cognitive neuropsychologists in the development of theories of normal function.

Language

The global impairment of language perception occurs in the rare syndrome of pure word deafness, in which the patient is able to perceive and comprehend environmental sounds and may understand the written word, but is unable to perceive language. Buchman *et al* (1986) compare three new cases of word deafness with 34 previously reported cases of various cortical auditory disorders, concluding that patients with word deafness, who have had formal testing of linguistic and non-linguistic sound comprehension and musical abilities, always demonstrated a more pervasive auditory agnosia. Tanaka *et al* (1987) report a case of a woman with bilateral temporoparietal lesion who developed pure word deafness with amusia. The clinical defect was limited to auditory modality although psychophysical data showed a defect in temporal resolution in visual and somatosensory modalities as well as in the auditory modality. The authors concluded that the auditory modality is much more dependent upon a temporal resolution than other sensory modalities.

The familiarity or frequency of a word is an important factor in word comprehension deficits. Studies of aphasic patients have shown that comprehension of familiar common words is more likely to be preserved than comprehension of less familiar words. Semantic categories of words have also been found to be a critical factor in word comprehension deficits. Warrington & McCarthy (1983) investigated a severe global dysphasic patient. Object names were comprehended significantly less well than flower, animal and food names. Analysis of response consistency and presentation rates provided evidence for a deficit in accessing particular semantic categories. Warrington & Shallice (1984) report four patients whose knowledge of certain categories of visual objects appeared to be selectively impaired. They all had much greater difficulty in identifying living things than inanimate objects. More recently, Warrington & McCarthy (1987) investigated the category specificity in a patient who was significantly more impaired in the comprehension of small manipulable objects than large man-made objects. These papers provide evidence for a fine-grain categorical organisation of knowledge systems.

A disorder of language output, characterised by a marked dearth of spontaneous speech without impairment of speech production *per se*, was first described by Lichtheim (1885). Luria (1970) termed this distinctive type of speech-output disorder 'dynamic aphasia'. Costello & Warrington (1989) report a single case study of a patient with a selective speech disorder which had all the hallmarks of a dynamic aphasia. The patient's ability to generate sentences from a verbal context was significantly worse than from a pictorial context. Although he could order a sequence of pictures, he had the greatest difficulty in ordering the constituents words of a sentence. These findings are discussed in relation to Luria's hypothesis that dynamic aphasia is due to an impairment of inner speech which provides the 'linear scheme of a sentence'. The authors argue the patient's difficulties in sentence construction arise at an earlier stage of verbal processing: it is the initial 'thought' or plan that is impoverished, not the ability to implement it.

Memory

Memory impairments encompass a wide range of deficits which may be both selective and dissociable. Baddeley (1976) provides a useful overview of memory. The first chapters focus on the early experimental studies by Ebbinghaus and Bartlett. Warrington & McCarthy (1986) bring together evidence for the various forms of memory deficit observed in patients with cerebral disease, and discuss their significance for the localisation of damage and their implications for theories of memory organisation. The amnesic syndrome in Korsakoff's psychosis exemplifies the global impairment of event memory in which the person typically presents with an anterograde and retrograde amnesia. Mair *et al's* (1979) detailed investigation of the memory defects in two patients with Korsakoff's psychosis found that the minimal lesion for the alcoholic Korsakoff amnesic state may be in the mammillary bodies or in the medial aspect of the thalamus. Butters & Cermak (1980) present an overview of the 'information-processing deficits' theory of amnesia, and contrast this with other theoretical approaches. Amnesic patients have been found to perform remarkably well on certain types of learning and retention tasks. To account for these differences, Warrington & Weiskrantz (1982) postulate a disconnection between the 'cognitive mediational memory system' and semantic memory system. The amnesic can learn simple stimulus-response (S-R) relationships as these do not require cognitive mediation.

There has been some debate as to whether memory for fact and memory for events are two relatively independent subsystems or whether they are on a continuum. A single case study reported by Warrington & McCarthy (1988) provides evidence for a discontinuity between these systems. The patient had a dense retrograde amnesia for both personal and public events, with relative preservation of vocabulary for famous names and visual vocabulary for famous faces. By contrast, Grossi *et al* (1988) present an interesting report of a patient with a selective memory loss for facts, with relative sparing of her memory for personal events.

More common are the selective memory impairments. Milner (1971) reviews the early studies that she and her colleagues conducted. Patients with left temporal-lobe lesions showed selective memory impairments for verbal material whereas patients with right temporal-lobe lesions showed deficits in their retention of non-verbal material. More recently, Warrington (1984) established modality-specific memory impairments in a large group of patients with well defined unilateral lesions, using recognition memory tests for verbal (words) and non-verbal (faces) material.

Visual perception

The integrity of the early visual information–processing mechanisms must be ascertained before diagnosis of higher-level perceptual deficits can be made. Deficits at this stage of visual processing are associated with a partial cortical blindness. Holmes' (1918) early reports of disturbances of vision and, in particular, visual disorientation are excellent. Warrington (1986) reviews the evidence for the fractionation of the visual functions (acuity, shape, colour, and location) and describes five patients who were considered to have made a partial recovery from an acute episode of cortical blindness. The findings show that these four functions are dissociable.

The classic paper by Lissauer (1890) described one of the earliest cases of visual agnosia. Lissauer distinguishes two stages in object recognition: that of conscious awareness of a sensory impression, apperception; and that of associating meaning with the content of this apperception, association. Taylor & Warrington (1971) report a case of associative agnosia. More recently, Warrington & James (1988) report three cases with all the hallmarks of an apperceptive agnosia. Warrington (1985) describes a model of object recognition derived from Lissauer's early classification, which includes two post-sensory categorical stages: the first involves visual sensory processing leading to perceptual categorisation; the second involves those processes that achieve semantic categorisation of an object. There are several good chapters in the book by Ellis & Young (1988) which cover object recognition and disorders at an earlier stage of visual processing, including colour, blindsight and visual location. Their chapter on face recognition, of which the most striking disorder is prosopagnosia, is of particular interest.

Spatial skills and related phenomena

De Renzi (1982) analyses disorders of spatial behaviour according to their level of breakdown, from impairment of scanning and direction of attention, to deficiency of spatial thought and memory. Material is drawn from three sources: clinical observations of brain-damaged patients; quantitative assessment of the performance of these patients and normal subjects; and anatomical, neurophysiological and behavioural findings in animals. Wide consideration is given to aspects of space exploration impairment, including unilateral neglect of space.

Bisiach & Luzzatti (1978) noted that neglect encompassed descriptions from memory of familiar surroundings. These authors asked two patients with visuospatial neglect to recall the buildings along two sides of the principal square of Milan, imagining in one case that they were facing in one direction and in the other case that they were facing in the opposite direction. In both trials the patients omitted to mention the buildings placed on their left, so that those recalled on one occasion were neglected immediately afterwards and vice versa. Neglect can extend to reading. Ellis *et al* (1987), in a detailed study of a patient who showed neglect dyslexia, suggested that this arises from a more general visual neglect. This position is, however, undermined by Costello & Warrington's (1987) report of a man with evidence of both a left-sided neglect dyslexia and right-sided visuospatial neglect. The occurrence of these two phenomena provides evidence of a dissociation of these forms of neglect.

Conclusion

Recently, neuroscientists have given increasing attention to the study of the human brain. This has resulted in a vast and growing body of knowledge as well as an advancement in the assessment, diagnosis and remediation of brain-related cognitive and affective disorders. I have selected a few areas, illustrating the contribution of neuropsychological research to our understanding of cognitive functions and behaviour.

References

Baddeley, A D. (1976) *The Psychology of Memory* New York Basic Books

Bisiach, E & Luzzatti, C (1978) Unilateral neglect of representation of space *Cortex*, **14**, 129–133

Buchman, A., Garron, D C., Trost-Cardamone, J., *et al* (1986) World deafness. one hundred years later *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 489–499.

Butters, N & Cermak, L. S (1980) *Alcoholic Korsakoff's Syndrome An Information-Processing Approach to Amnesia* New York. Academic Press.

Costello, A. de L & Warrington, E. K. (1987) The dissociation of visuo-spatial neglect and neglect dyslexia *Journal of Neurology, Neurosurgery and Psychiatry*, **50**, 1110–1116.

—— & —— (1989) Dynamic aphasia· the selective impairment of verbal planning *Cortex*, **25**, 103–114.

De Renzi, E. (1982) *Disorders of Space Exploration and Cognition* Chichester Wiley.

Ellis, A W , Flude, B M & Young, A W (1987) Neglect dyslexia and the early visual processing of letters in words *Cognitive Neuropsychology*, **4**, 439–464.

—— & Young, A. W (1988) *Human Cognitive Neuropsychology* London Lawrence Erlbaum

Grant, I & Adams, A. M. (1986) *Neuropsychological Assessment of Neuropsychiatric Disorders*. New York· Oxford University Press

Grossi, D , Trojano, L., Grasso, A , *et al* (1988) Selective "semantic amnesia" after closed-head injury *Cortex*, **24**, 457–464

Heilman, K M & Valenstein, E. (eds) (1985) *Clinical Neuropsychology* (2nd edn). New York: Oxford University Press

Holmes, G (1918) Disturbances of visual orientation *British Journal of Opthalmology*, **2**, 449–468

Lichtheim, L. (1885) On aphasia *Brain*, **7**, 433–484.

Lissauer, H (1890) Ein Fall von Seelenblindheit nebsteinen Betrag zur Theorie derselben. (Trans M. Jackson, 1988) *Cognitive Neuropsychology*, **5**, 157–192

Luria, A R. (1970) *Traumatic Aphasia*. The Hague Mouton.

Mair, G P , Warrington, E K & Weiskrantz, L (1979) Memory disorder in korsakoff's psychosis. a neurological and neuropsychological investigation of two cases. *Brain*, **102**, 749–783.

Milner, B (1971) Interhemispheric differences in the localisation of psychological processes in man. *British Medical Bulletin*, **27**, 272–277.

Robbins, T W. & Cooper, P J. (eds) (1988) *Psychology for Medicine*. London. Edward Arnold

Shallice, T. (1988) *From Neuropsychology to Mental Structure* Cambridge· Cambridge University Press

Tanaka, Y., Yamadori, A. & Mori, E (1987) Pure word deafness following bilateral lesions: a psychophysical analysis *Brain*, **110**, 381–403.

Taylor, A & Warrington, E. K (1971) Visual agnosia. a single case report. *Cortex*, **7**, 152–161

Warrington, E K (1984) *Recognition Memory Test* Windsor· NFER.

—— (1985) Agnosia. the impairment of object recognition In *Handbook of Clinical Neurology* (ed. J. A. M Frederiks) Amsterdam Elsevier

—— (1986) Visual deficits associated with occipital lobe lesions in man *Pontificiae Academiae Scientiarum Scripta Varia*, **54**, 247–261

—— & James, M. (1988) Visual apperceptive agnosia a clinico-anatomical study of three cases. *Cortex*, **24**, 13–32

—— & McCarthy, R. A. (1983) Category-specific access dysphasia *Brain*, **106**, 859–878

—— & —— (1986) Disorders of Memory. In *Disease of the Nervous System*, **2** (eds A K Asbury, G. M. McKhann & W I McDonald). London Heinemann Medical Books

—— & —— (1987) Categories of knowledge further fractionations and an attempted integration. *Brain*, **110**, 1273–1296

—— & —— (1988) The fractionation of retrograde amnesia *Brain and Cognition*, 7, 184–200.

—— & Shallice, T. (1984) Category-specific semantic impairments *Brain*, **107**, 829–853.

—— & Weiskrantz, L (1982) Amnesia: a disconnection syndrome *Neuropsychologia*, **20**, 233–249.

Angela Costello, FRN, BSc, MSc (ClinPsych), *Senior Clinical Neuropsychologist, Brook General Hospital, London SE18 4LW*

British Journal of Psychiatry (1990), 157, 464–473

Book Reviews

Editor: Sidney Crown

Motivation and Explanation: An Essay on Freud's Philosophy of Science. By NIGEL MACKAY. Madison, Connecticut: International Universities Press. 1989. 254 pp. $27.50.

Jaspers' celebrated distinction between causal and meaningful connexions gave expression to one of psychiatry's fundamental dilemmas: the apparent opposition between the search for causes of psychological disorder, and the discernment of its meaning. The distinction between cause and meaning came from the cultural sciences of the turn of the century, and the issues it raises remain prominent in contemporary philosophical debate. Causal explanations plausibly have to invoke scientific laws concerning material processes, while explanations of behaviour in terms of meaningful mental states invoke reasons, motivation, purpose, etc. If the two types of explanation are regarded as essentially distinct and mutually exclusive, there are broadly two options. One option, traditionally associated with materialism and reductionism, is to deny the validity of meaning. The other is the hermeneutic approach, which affirms the autonomous validity of the science of meaning, hence forcing a rupture between it and natural science. In psychiatry these fundamental issues have been battled out largely in relation to psychoanalytic theory. The theory has been criticised from outside, for being preoccupied with meanings as opposed to causes. But further, the debate has arisen within psychoanalysis itself. Here the focus has been the apparent tension between Freud's scientific, causal metapsychology, and the clinical concern with meaning. The hermeneutic readings of psychoanalytic theory popular in the 1960s and 1970s rejected the former in favour of the latter.

Mackay's book is concerned primarily with this debate within psychoanalysis. The author argues that Freud's metapsychology, in which the concept of motivation is central, is a legitimate scientific theory, and one compatible with material causation. In this argument Mackay draws on contemporary realist philosophy of science and philosophy of mind. He argues convincingly that the metapsychology encompasses what is essential to meaningful explanations. In this way the schism between psychoanalytic metatheory and clinical theory, in general that between causality and meaning, is broken down. In the course of the argument the author examines in detail Freud's own philosophy of science and mind, bringing out its frequently underestimated depth and subtlety. Mackay successfully brings contemporary philosophy to bear on these important issues. The book can be recommended to the reader interested in the logic of psychological explanation in general, and of psychoanalytic explanation in particular.

DEREK BOLTON, *Senior Lecturer, Psychology Department, Institute of Psychiatry, London*

The Problem of Loss and Mourning: Psychoanalytic Perspectives. Edited by DAVID R. DIETRICH and PETER C. SHABAD. Madison, Connecticut: International Universities Press. 1990. 499 pp. $55.00.

William Shakespeare might have been preparing an introduction to this book when he wrote:

"But if the while I think on thee dear friend,
All losses are restor'd and sorrows end."

Sigmund Freud was the first important analyst to study grief, and then chiefly as a key to understanding melancholia. Grief may follow losses or anticipate them. Losses leading to grief may be intrapsychic and difficult for the observer to understand or empathise with. They may be overt, massive and with universal impact, as in the case of the Holocaust. The sheer variety of possible grief-inducing losses known to mental health workers and written about is expanding daily. Limbs, breasts, pets, property, self-esteem, and so on, have been added to parents, spouses, and children.

In more recent years we have begun talking about the psychotherapy of the dying, helping others to face the ultimate loss, that of the self. For too long this was a taboo subject, most likely because of the anxiety it caused in carers and the consequent threat it presented to the universal subconscious belief in one's own immortality. We all actively practice denial all of the time when it comes to that dreaded subject, even if we write or speak to the contrary.

Victimology, that other new branch of psychological thinking, has been highlighted by the study of Nazi atrocities and by the effects of kidnapping, rape, etc. on the survivor. A little studied aspect, which is only recently receiving attention, is the handling of guilt by individual perpetrators and even whole nations. The study of the post-traumatic stress disorder has guided researchers into the study of transgenerational grief. Dietrich & Shabad, both American psychologists, have neatly brought together 21 authors and 16 chapters to give the reader a comprehensive view of present psychoanalytical thinking on the monumental subject of mourning and the losses that invoke it. We are reminded that we all experience multiple losses. While the effects of such losses may be extremely negative, the result may also be a flowering of creative talent. How many great paintings and symphonies owe their existence to their authors' sense of loss? Such is the theme in much Celtic singing.

The book looks at the interesting possibilities for harmful experiences in the children of dead, divorced or single parents. As I read these pages I was struck by my memories of the protestations of good parenting ability made recently on television by homosexual couples rearing a child. The authors acknowledge the relative lack of knowledge concerning grief in the elderly. Most of our theories come from the study of the young.

The book varies widely in its readability. However, the editors neatly wrap the text between a helpful introduction and an accurate summary chapter. This book is essential reading for all students of grief, even if the reader might feel that some of the arguments are based on narrowly collected data.

BRIAN O'SHEA, *Consultant Psychiatrist, Newcastle Hospital, Greystones, Republic of Ireland*

Group Psychodynamics: New Paradigms and New Perspectives. Edited by DAVID A. HALPERIN. London: Wolfe Medical Publications. 1989. 331 pp. £38.50.

This American text has three parts, 21 chapters and 23 different authors.

Part one covers historical and theoretical perspectives. The focus is mainly on transference and countertransference and the effects of the group leader on the development and psychodynamics of groups. The theoretical discussions tend to lack clinical illustrative material and do not make easy or interesting reading.

In part two the focus is on 'new' populations and includes chapters on group therapy with AIDS patients, cocaine abusers, women with pre-menstrual syndrome, parents of children with chronic illness, process groups for psychiatrists in training and others. As there are many processes common to all groups, whatever the particular characteristics of the individual members, there is a great deal of repetition in this section and very little that is, strictly speaking, new. Even the chapter on group therapy with AIDS patients reveals no genuinely novel insights, although it does warn of the need to protect group members with AIDS encephalopathy from too much confrontation by other members, and points out that members who appear 'inappropriately passive' might well be suffering from the psychomotor retardation of AIDS encephalopathy.

Psychiatrists are the 'new' population discussed in the chapter on process groups in training programmes for 'residents'. Precisely what constitutes a process group is defined and contrasted with a therapy group. The particular problems attached to running a process group are usefully discussed.

Yet again in part three there is much repetition. Three of the seven chapters deal with adolescents, and a further chapter on group therapy with girls without fathers is a rich mixture of myth, self psychology and case material from latency age patients. One chapter on group therapy with geriatric populations reveals that elderly people can change to a surprising degree, but not surprising is the observation that the themes of narcissistic loss and deprivation are dominant within the group. Other chapters on group therapy with Soviet immigrants and short-term group psychotherapy with the functionally ill appear out of place.

In conclusion, although there are some very interesting contributions, this book suffers from the effects of multi-authorship, much repetition, divergent styles (in this particular case, an uneasy mix of research findings and psychoanalytic theory), and little to tie the chapters together. At the price of £38.50, I would recommend that your library should borrow it on request rather than buy it.

STEPHEN P. REILLY, *Consultant Psychiatrist with Special Responsibility for Psychotherapy, Bootham Park Hospital, York*

Theatres of the Body: A Psychoanalytic Approach to Psychosomatic Illness. By JOYCE MCDOUGALL. London: Free Association Books. 1989. 183 pp. £10.95 (pb), £25.00 (hb).

Originally from New Zealand, McDougall has worked for many years in Paris where she is a training analyst of the Paris Psychoanalytic Society. This current book is a sequel to her *Theatres of the Mind*, in the latter part of which, having dealt with the 'neurotic theatre', followed by the 'psychotic', 'transitional' and 'narcissistic' theatres, she found herself struggling with a theatre that she named, provisionally, 'the psychosoma on the psychoanalytic stage'. This book elaborates that theme, one stated aim being "to study, from a psychoanalytic viewpoint, the unconscious significance of psychosomatic manifestations and to examine the extent to which these are linked to the vicissitudes involved in becoming a person and to failures of the internalisation processes by which individual identity is constructed" (p. 48).

She traces the evolution of concepts in this area, in particular the French concept of '*pensée operatoire*' (here translated as 'operatory thinking' rather than the more usual 'operative') and the Boston concept of 'alexithymia'. She now finds shortcomings in both of these and prefers her own concept of 'disaffectation'. These all seem to be different ways of saying something similar about being cut off from feelings, and I am slightly resistant to her tendency to introduce fresh neologisms, of which there are several here (e.g. 'transference osmosis').

In the clinical illustrations she is, as usual, an excellent story-teller, although at times with a rather breathless style, full of exclamation marks. This is most evident in the brief biographical vignette of her New Zealand family origins. However, her admirers will not be disappointed. Others may wonder if these case histories represent a golden pre-scientific age of psychoanalysis, full of excellent natural history descriptions but lacking either the rigour which some analysts may seek or the scientific evidence which readers of this Journal might demand. Some may also wonder at the *ingenue* style (e.g. "She manifested severe obsessional symptoms, which I had been taught were a 'good indication' for psychoanalytic treatment"; p. 92), or at the length of some of the analyses reported, often second analyses by the author of 5–9 years on top of previous analyses of 5–12 years. At least she asks rhetorically: "Why do we accept in analysis those whose aim is to resist the psychoanalytic process . . ." (p. 116); and also attempts an answer: "No doubt we are sometimes motivated by the narcissistic wish to succeed where others have failed!" (p. 117, her exclamation mark). The book ends with rather a rushed precipitate half-page conclusion as if in a hurry to meet a deadline.

JONATHAN PEDDER, *Consultant Psychotherapist, Maudsley Hospital, London*

The Psychoanalytic Core: Essays in Honour of Leo Rangell. Edited by HAROLD P. BLUM, EDWARD M. WEINSHEL and ROBERT F. RODMAN. USA: International Universities Press. 1989. 536 pp. $60.00.

As the subtitle indicates, this book is composed of a series of chapters written in honour of Leo Rangell, a most distinguished North American psychoanalyst whose scholarly, systematic and extensive contributions to the psychoanalytic literature are recognised as outstanding. His work has had a profound influence on many analysts, and many of his numerous articles and books, published over a period of 45 years, have become classics. He is in the unique position of having twice been awarded the clinical essay prize of the British Psycho-Analytical Society.

The fact that Rangell has shown profound interest in a wide range of areas within psychoanalysis is reflected in the *Festschrift*. The many outstanding psychoanalysts who have contributed give the reader a chance to reflect on most of the theoretical, clinical and applied aspects of psychoanalysis. The book begins with the three papers directly concerned with Rangell's work and life (by Blum, Arlow and Rodman). Following this, a number of stimulating chapters discuss central psychoanalytic issues such as dreams and neurotic symptoms (Brenner), feelings of loss and inconsolability (Weinshel), perversions (Kernberg and Stoller), and pathogenic aspects of the primal scene (Harley).

In 1980 Rangell published an influential book entitled *The Mind of Watergate: An Exploration of the Compromise of Integrity*. This, together with a series of papers written by Rangell at that time, illustrate his psychoanalytic reflections on this topic. A number of contributors, among them Rodman, Arlow, Blum, Schengold, Chasseguet-Smirgel, Glenn, Pearl King, Grinberg, Treurniet, and Lebovici, have added their theoretical, clinical and applied contributions to the understanding to the role of morality and integrity in mental functioning. Rangell's interest in all aspects of psychoanalysis has been marked by a willingness to extend and enlarge concepts if necessary, while at the same time remaining firmly rooted in basic psychoanalytic ideas. Thus it is no surprise that he developed a great interest in the work of Anna Freud, an interest which is reflected in a number of contributions in the *Festschrift*; perhaps most clearly in Yorke's chapter on "Freud in a second generation – some personal reflections". In addition to this, the contributions of Wallerstein, Van Dam, Galenson, Fields, Etchegoyen, Limentani, Jucovy, and Moses complete a most scholarly work.

This volume is of substantial intellectual breadth and is indeed a fitting tribute to an exceptional psychoanalyst.

ANNE-MARIE SANDLER, *Psychotherapist, British Psycho-Analytical Society, London*

The Management of Children with Emotional and Behavioural Difficulties. Edited by VED P. VARMA. London: Routledge. 1990. 176 pp. £10.95.

This handy paperback is written for students in the various fields of the helping professions. The contributors include an educationalist, a social worker, two psychotherapists, two psychologists and three psychiatrists. Their differing perspectives enable the reader to obtain a fairly comprehensive picture of the available models and management approaches to children's emotional and behavioural difficulties. The point is made that individual symptoms and types of behaviour need to be assessed by relating them to the social and family contexts within which they occur. Most chapters are clearly written and contain much useful although basic information. Psychodynamic and behavioural approaches are particularly well represented. The family systems model, on the other hand, is insufficiently discussed. In summary, this is a useful multidisciplinary introductory text for beginners.

K. ASEN, *Consultant Child Psychiatrist, Barnet Child Guidance, London*

On Jung. By ANTHONY STEVENS. London: Routledge 1990. 302 pp. £15.95.

The first section of this book is a basic outline of Jung's analytical psychology, giving accessible and understandable accounts of Jung's thoughts on personal equation, the unconscious, metapsychology and personal development. Sections two and three trace life from childhood through adolescence to early maturity, mid-life transition, middle age, late-life transition and late maturity using Jung's own personal life history to illustrate his theories.

Stevens presents the book to us "using classical concepts, within a developmental framework while at the same time not neglecting the creative importance of imagination which is the particular emphasis of the archetypal school".

Throughout, the author quotes from and makes reference to a varied range of people, from the ancient Greek philosophers Plato, Aristotle and Socrates, through Shakespeare and Darwin to the great 19th-century dramatists Checkov, Ibsen and Strindberg. Others include the philosopher Nietsche and those European explorers of the unconscious mind Caris, Schopenhauer and Von Schubert, as well as the obvious doyens Freud, Klein and Adler, and his contemporaries Storr and Samuels.

The book flows, and I found two of its early chapters on "Childhood" and "Adolescence" particularly clear, and interesting. In one of the final chapters, "Late Maturity", Stevens deals with Jung and alchemy; although informative, this chapter is long-winded and I found the equation with Jung's personal life and psychology to be unclear. The conclusion is warm, lucid and brings the book to a positive, enjoyable and satisfying end.

Stevens gives a clear and concise exposition of basic Jungian concepts from the archetype and collective unconscious to the anima and animus as well as a comprehensive and searching look at Jung the man, his life, and his work.

IAN O'REILLY, *Editorial Assistant, British Journal of Psychiatry*

Closely Observed Infants. Edited by LISA MILLER, MARGARET RUSTIN, MICHAEL RUSTIN and JUDY SHUTTLEWORTH. London: Duckworth. 1989. 221 pp. £10.95.

This book is essentially an account of what we can learn from babies of their early encounters with the world, and of their developing skills and emotional growth. It also describes some of the infinite variety of responses by family members to the newcomer.

Broadly, the technique of infant observation comprises regular weekly visits to a family, lasting about an hour, and continuing over two years if possible. The observer is encouraged: (a) to intrude as little as possible into the situation; and (b) to record as fully as possible what takes place, and to report this in a small seminar group, where its significance can be studied The observer first learns to record accurately events of which the underlying significance is not understood or sought for at the time. The richness of the inner life of babies will be made accessible to the participants subsequently and often dramatically. It almost invariably provides a fruitful experience, increasing the understanding not only of babies, but also of parents and others closely involved

Infant observation is a growth industry, and consequently it is possible that the quality of the work may deteriorate in general. However, this is not the case here where there is an extremely high standard, both in the six reports of infants observed, and in the additional chapters. Miller gives a clear general introduction; Rustin writes sensitively on the problems confronting observers, both external and personal; Rustin gives a comparison of the method of psychoanalytically-oriented work with those of other disciplines; and Shuttleworth contributes a detailed and scholarly account of child development. The core of the book, however, remains the six case studies. These must make absorbing reading for any clinician working with children, and may well prove of great help to all workers in the field of psychiatry.

ELINOR WEDELES, *Member of the British Psychoanalytical Society*

Sleep and Alertness: Chronobiological, Behavioral and Medical Aspects of Napping. Edited by DAVID F. DINGES and ROGER J. BROUGHTON. New York: Raven Press. 1989. 346 pp. $111.50.

Napping might seem an unlikely topic for a book, but this collection of articles by experts in various aspects of sleep turns up a surprising amount of information. Napping is defined as "any sleep period with a duration of less than 50% of the average major sleep period of an individual."

After Tobler reviews napping in mammals, Webb charts the development of napping in humans from the biphasic sleep pattern at age six months to the single afternoon nap at the end of the first year followed by a decline during childhood but a full return after puberty. Thereafter, there is a linear increase in napping with advancing age. Webb favours the 'MD and E' explanation (i.e. only mad dogs and Englishmen go out in the noonday sun). Put more scientifically, napping has developed as a behavioural control to avoid the torrid heat of midday.

Carskadon discusses the relationship between age and sleepiness, concluding that the human brain is programmed for a siesta and recommends college students to take a planned midday nap to prevent falling asleep during afternoon lectures. Broughton then reviews chronobiological aspects of sleep and napping and concludes that there are two distinct 'gates' in the 24 hours when the transition from sleep to waking is facilitated, a primary nocturnal and a secondary mid-afternoon period. These views are confirmed in some experimental observations by Lavie and the evidence from napping in time-free environments discussed by Campbell & Zulley.

Stampi reviews the difficulty in maintaining performance in situations of ultra-short sleep schedules, including some fascinating observations on the crews of ocean-racing yachts. Dinges provides a comprehensive account of the anatomy, physiology and psychology of the human nap and concludes that naps have beneficial effects on mood and performance once the immediate post-sleep inertia has dissipated. Akerstedt *et al* cover the effects of shift-work and Naitoh & Angus those of prolonged work. Webb & Dinges discuss cultural aspects of napping and the siesta and Broughton reviews pathological sleep disorders. In a final integrative chapter, the editors sum up the book with the comment that the study of napping might well unlock the mystery of sleep itself.

This book, which I found immensely reassuring, will appeal to all interested in sleep research.

KENNETH DAVISON, *Consultant Psychiatrist Emeritus, Newcastle upon Tyne*

Cognitive Behavioural Therapy for Psychiatric Problems: A Practical Guide. Edited by KEITH HAWTON, PAUL M. SALKOVSKIS, JOAN KIRK and DAVID M. CLARK. Oxford: Oxford University Press. 1989. 456 pp. £40 (hb), £15 (pb).

Cognitive therapy has been very much the fashionable psychotherapy for the last decade. This handbook chronicles the use of cognitive behavioural techniques in a wide variety of different psychological disorders. It would appear that the history of new psychotherapies is very much like that of new drugs. They are first used by a few enthusiasts, tested, and introduced to the market for use with a specific group of conditions in circumscribed clinical settings. Next, they are taken up more widely and begin to be used in a multitude of different settings and different situations for a wide variety of conditions before disillusionment sets in. Finally, they either disappear without trace or find their place in the market along with other treatments. At present, it seems that one can use cognitive behavioural therapy for almost anything, and it has certainly moved a long way from its origins as a highly structured, time-limited, specific treatment for depressive disorders. It has become less structured, more open-ended, and used for longer-term treatment and for more challenging behaviours such as personality disorder. Presumably, time will tell.

One refreshing thing about this book is that it presents essentially the 'British' view of cognitive therapy as originally practised in Oxford and Edinburgh, and now in many centres around the UK. Over half the authors are associated with the Oxford department and this indicates the breadth of expertise that Oxford has in this area. One essential characteristic of British, or perhaps more correctly, European, cognitive behavioural therapy seems to be its close adherence to the principles outlined by Beck in the 1970s and early 1980s. Despite this 'conservatism' it is this school that has advanced the treatment into many other areas such as hypochondriasis and obsessive–compulsive disorder. This book has chapters on assessment, anxiety and panic disorders, phobic disorders, obsessional disorders, depression, somatic problems, eating disorders, chronic psychiatric handicaps, marital problems, sexual dysfunction and problem-solving. It is therefore much more wide ranging than its competitors. Unlike many books that call themselves handbooks or practical guides, this one does live up to its name since it *does* tell you how to do it. There are no weak chapters, all being written to a uniformly high standard, but those who are looking for a new cognitive perspective to the treatment of sexual dysfunction or problem-solving will be disappointed. These chapters are really good old-fashioned behaviour therapy. In a sense I see this as a plus. The authors have resisted the trend to stick the adjective 'cognitive' on the front of every type of behaviour therapy just because the patient has cognitions or thinks.

One of the most usual comments heard from clinicians of all backgrounds when cognitive behaviour therapy is described to them is: "oh well, that's what I do anyway". After many hours of watching video tapes of clinical interviews I have never seen any evidence of this, and, in fact, many so-called cognitive behaviour therapists do precious little cognitive behaviour therapy either. This book will help those who are already practising to improve their technique and it will certainly help those who are just beginning in this area. There are several other books on the market that cover similar topics but this has to be seen as the market leader. It is easy to read, uniformly well written and the most comprehensive of the practical handbooks available. I fully recommend it.

CHRIS FREEMAN, *Consultant Psychotherapist, Royal Edinburgh Hospital, Edinburgh*

Handbook of Phobia Therapy: Rapid Sympton Relief in Anxiety Disorders. Edited by CAROL LINDEMANN. New Jersey: Jason Aronson. 1989. 456 pp. $45.00.

The book opens with a brief historical overview of aetiological theories and their influence on the development of treatments for phobic disorders. Although the next chapter covers classification, outlining the DSM–III system, important nosological issues are not critically discussed. The first section of the book includes another six chapters which describe the clinical features of agoraphobia, obsessive–compulsive disorder, childhood and flying phobias, and evaluative anxiety disorders of a generalised and specific nature. A detailed account of management follows the description of each condition (apart from evaluative anxieties), incorporating behavioural and some cognitive approaches. The chapters on agoraphobia and compulsive ritualisers provide excellent accounts of treatment.

The second part of the book consists of several chapters describing strategies and techniques that can be used to aid anxiety reduction and to assist exposure (e.g. breathing control in hyperventilators, education of patients about anxiety and coping tactics). Two further chapters describe marital and interpersonal therapies which can be helpful as adjuncts to exposure in some cases. The book ends with one chapter each on the psychoanalytic approach to phobias and drug treatments of anxiety disorders.

This is a multi-authored book and there is some repetition. Some important topics, such as the treatment of social phobia, are omitted, but there is a critical discussion and evaluation of the various treatment approaches described in the book. In addition, as much of the book is devoted to the behavioural approach, an introductory chapter setting out behavioural principles (analysis, goals, negotiation, measurement, evaluation and feedback) would have been useful, instead of these being scattered throughout the book.

The book has a few excellent chapters but, in my opinion, is not in itself a complete handbook. It is suitable for libraries rather than the individual. Furthermore, in these days of limited funds, it is rather expensive.

GERALDINE O'SULLIVAN, *Lecturer. Department of Experimental Psychopathology, Institute of Psychiatry, London*

Clinical Approaches to Violence. Edited by KEVIN HOWELLS and CLIVE R. HOLLIN. West Sussex: Wiley & Sons. 344 pp. £35.95.

This book aims to present a cognitive and behavioural analysis of the causes of violence and to provide a basis for planning the treatment of people who have been violent. It looks at violence in a number of settings, in particular within the home and various institutions.

'Clinical' in the title of the book means paying "primary attention to the psychological characteristics of the individually violent person". The editors do not attempt to include the relationship between mental illness and violence (another book in this series will cover the topic). Nevertheless, although it is aimed primarily at probation officers and psychologists, sections of the book are of interest to psychiatrists.

The most successful chapter is that on sexual violence. Here, the authors unite research evidence into a coherent theoretical framework which then serves as a basis for a treatment model used by the authors. They describe its application both in general terms and with an account of one case and go on to describe the evidence that their treatment has some effect on recidivism. Unfortunately, none of the other chapters combine research work with practical experience of treatment in such a comprehensive fashion.

The chapter of most obvious potential relevance to psychiatrists is that on violence in psychiatric hospitals. This describes the research that has been done in the field and points to how little is known. This chapter is not in fact about the individuals who commit violence on a ward but about the staff responses. Its main argument is that the standard means of managing violence on the ward can reinforce it, but the strength of evidence used to support this argument is weak. For example, it argues that neuroleptic medication has "been found to promote violence", but this sweeping claim is supported by one reference which turns out to be a single case study.

The central chapters review the literature on domestic violence. These sections go beyond the declared scope of the book in describing victim characteristics and more general social factors. This widening of scope seems to be because the authors do not have all that much to say on the perpetrators of violence, and the section on treatment (of the perpetrators) is vague and does not appear to be based on any practical experience.

Overall, the book is a useful source of information on the causes of violence within the individual but less useful as a guide to treatment.

MARK SWINTON, *Research Worker, Department of Forensic Psychiatry, Institute of Psychiatry*

Diagnosis and Treatment of Senile Dementia. Edited by M. BEGENER and B. REISBERT. Berlin: Springer-Verlag. 1989. 389 pp. £45.50.

This book is made up of papers given by a wide range of authors at a workshop in Vienna in 1989. The preface states that it intends to be "a comprehensive overview of current knowledge in diagnosis, prevention and treatment of senile dementia". While there is some basis to this statement as far as diagnosis and prevention are concerned, the section on treatment is certainly not comprehensive.

The book is not concerned with the care and management of people with dementia other than in relation to drug treatment, and with regard to this, only one chapter considers the possible therapeutic role of drugs affecting monoamine, cholinergic, neuropeptide and gabanergic transmission. The last 120 pages of the book are exclusively given over to papers about calcium channel blockers and to one compound in particular. The workshop was sponsored by a drug company and I can only assume, perhaps incorrectly, that there is a connection here. The importance of this section of the book will depend on whether this group of drugs is shown in the long term to be of any value in the treatment of senile dementia.

The rest of the book is of more general interest and is quite readable. The initial chapters are concerned with epidemiology and with factors associated with dementia. Later chapters deal with differential diagnosis, psychological assessment of dementia, and clinical and diagnostic elements including a discussion of males and positron emission tomography scanning. There is a particularly interesting chapter on the treatment of depression in the elderly and, in general, the chapters on differential diagnosis were the most interesting.

Despite the limitations of the book mentioned earlier, this would be a worthwhile addition to a hospital library.

Tony Roberts, *Senior Registrar in Psychiatry, Bangor*

Alzheimer's Disease. Treatment and Long-Term Management. Edited by Jeffrey L. Cummings and Bruce L. Miller. USA: Marcel Dekker. 1990. 416 pp. $125.00 (USA and Canada), $150.00 (all other countries).

Increasing concern for the biomedical importance and social consequences of Alzheimer's disease (AD) is leading to an abundance of publications on this subject. This latest offering comes from California, and contains 25 papers contributed by 34 authors, 31 from North America and three from Europe. A range of topics is covered, grouped under five headings: "Introduction" (a single paper on clinical diagnosis); "Disease specific therapies"; "Treatment of behavioural symptoms", "Long-term care"; and finally "Future treatment directions".

While the price causes a sharp intake of breath, what is most impressive about the content is the appropriateness of the mix and the consistently high quality. All contributions are carefully researched, coherently presented, and well referenced. Predictably, familiar territory is covered: clinical diagnosis; incontinence; recognition and management of superimposed medical conditions; neuroleptics; antidepressants; cholinomimetics, and the ergoloid mesylates. However, the fact that stories can improve with re-telling is supported by these papers, and Ouslander's offering on incontinence is the best I have yet read.

Two other papers in the long-term care section are also exceptional: Morishita on wandering behaviour and McEvoy on behavioural treatment. By contrast, the paper on rehabilitation is disappointing and contains no reference to cognitive rehabilitation (reality orientation and reminiscence). In the USA, AD represents a financial catastrophe for most affected families (Read on community resources), while locally available and comprehensive assessment, treatment, rehabilitation and care programmes, rapidly becoming commonplace across the UK, are seldom to be found. However, Congress is already putting millions of dollars into AD research (67 million in 1987 alone) and may well heed the pleas of senior citizens and promote sounder and more affordable care systems as proposed by Jazwiecki in a chapter on future treatment directions. The spin off from already funded biomedical research is to be found in chapters on cholinergic enhancement strategies, genetic engineering, neuropeptides, amyloid angiopathy, and intra-cerebral grafting and neurotrophic factors: all superbly presented and, thankfully, understandable.

Overall, this is a very impressive book which should be read by clinicians working in multidisciplinary geriatric psychiatry teams, and by those who would like to research some aspect of dementia but don't know how to start – they will find plenty of suggestions.

In summary, the book is expensive, but the best usually is!

Garry Blessed, *Emeritus Consultant Psychiatrist, MRC Chemical Neuropathology Unit, Newcastle General Hospital, Newcastle Upon Tyne*

Depressive Disorders and Immunity (Progress in Psychiatry Series). Edited by Andrew H. Miller. Cambridge. Cambridge University Press. 1989. 189 pp. £17.50.

This book records the substance of a symposium organised by the American Psychiatric Association in 1986, although the format and references reveal that the papers have been substantially updated. Unlike many published proceedings of meetings, the publication of this monograph is fully justified. Firstly, its subject is essentially multidisciplinary and thus it was worthwhile encapsulating the views of the psychiatrists, immunologists and neurobiologists. Secondly, the interactions between stress, the immune system and psychiatric disorders have been the stuff of considerable mythology and dubious clinical applications, so an authoritative review was certainly timely.

The book contains eight contributions on the theme of depressive disorders and immunity. Fortunately, the editor is a polymath who, by appearing as an author on half of these contributions, has ensured continuity of style and an absence of repetition. Above all, the book is free of excessive jargon, and the technical terms peculiar to each discipline are properly explained. This is an indispensable virtue in a book designed mainly to introduce psychiatrists to concepts drawn from unfamiliar fields. In principle, these chapters deal with the physiological basis of interactions between the immune and nervous systems and with the clinical observations obtained with 'neuroimmunopharmacological' techniques in depressive illnesses. Only the final chapter is indisputably contentious; the history of research into many diseases with psychiatric manifestations from systemic lupus erythematosus to the post-viral fatigue syndrome is strewn with the wreckage of viral hypotheses, based solely on the pattern of antiviral antibody titres.

This is in no sense a didactic compilation. In the spirit of many good symposia, it provides food for the imagination rather than information by rote. For example, it has long been apparent that neurobiologists and immunologists face similar problems, and not least the need to understand the basic property of memory shared by the immune and central nervous systems. Thus, many immunologists may be unaware of the extraordinary experiments in which saccharine conditioning in lupus-prone mice induced immunosuppression and amelioration of disease; these experiments are discussed in the chapter on "Neural-immune interactions". Clinical psychiatrists too will appreciate still better the wide gulf between documenting abnormalities in lymphocyte function in depressed or stressed individuals and attributing aetiological significance to such observations. The lymphocyte may look like an orderly beast in the neat, stylised drawings of this and other texts, but in reality it bristles with antennae like a recirculating satellite, sensitive to every nuance in the changing biological environment.

A. M. DENMAN, *Honorary Consultant Physician, Clinical Research Centre and Northwick Park Hospital*

Mental Health Law in Context: Doctors' Orders? By MICHAEL CAVADINO. Aldershot: Dartmouth Publishing. 1989. 189 pp. £28.50.

There is little new in this legal researcher's look at how the 1959 Mental Health Act operated at a large (Victorian) mental hospital, whimsically called 'Fardale' by the author. The findings, based on data collected in the mid-1970s, are somewhat dated, as is the author's discussion. Fifteen years on we have new legislation, anti-psychiatry is *passé* and consumers criticise non-intervention rather than over-intervention.

The first part of the book reviews anti-psychiatry literature with an emphasis on the sociology of institutions and labelling theory. It lacks critical depth and has omissions that would be obvious to a psychiatrist in his/her first year of training. Rampton Hospital (pre-Boynton) is referred to as though it epitomised contemporary psychiatric practice. Research findings are reported in the second section and in more detail in an appendix. Cavadino worked on the wards during the study period and interviewed 96 consecutive admissions, questioned psychiatrists and social workers involved in 25 admissions (mostly compulsory) and spoke to a sample of 71 other in-patients. Unusually for this wort of work the data are statistically analysed. What did he find? Compulsory admissions consisted mainly of violent schizophrenic men; some patients resented their detention, others were passive; some psychiatrists used the Mental Health Act more than others; some section papers were wrongly completed; and sometimes psychiatrists and social workers disagreed. Fardale, it seems, is like everywhere else.

Yet from these unremarkable findings, the author recommends a rewrite of current legislation. It is difficult, however, to take his proposals seriously. emergency admission should only occur where there is immediate danger of death or severe injury; long-term detention is not really justifiable at all and should be dealt with under the criminal law. This book lacks balance and shows limited understanding of the effects of major mental disorder on patients and their relatives. Cavadino asks "Are mental health laws doctors' orders?"; if he means "Is the doctor's role pivotal in compulsory admission?", the answer is an unashamed "yes". That is what he is trained for and that is what he is paid for

DEREK CHISWICK, *Consultant Forensic Psychiatrist, Royal Edinburgh Hospital*

No Fault Compensation in Medicine. Edited by RONALD D. MANN and JOHN HAVARD. London: Royal Society of Medicine Services. 1989. 290 pp. £20.00.

This book comprises both the papers given and the transcripts of discussions that took place at a joint meeting between the Royal Society of Medicine and the British Medical Association on the above topic in January 1989. All interested parties are represented as speakers: doctors, lawyers, representatives of victims of medical accidents and representatives of the insurance industry.

The first few papers describe existing and similar schemes in Sweden, New Zealand and Finland. These were very detailed papers; it would have been helpful, however, to have had a rough monetary equivalent given for Swedish kroner. The New Zealand paper highlights the difficulty of deciding, in law, what a 'medical accident' is and how it should be defined, particularly in relation to psychological suffering. It also emphasised the importance of quality control within medical treatment; an issue, relating to the prevention of medical accidents, which came up many times in discussion. The discussion also pointed out the political dimension – in countries with high government spending on social security and high taxation, accident compensation schemes may work better because the amount of compensation will be proportionally lower.

A particularly good paper was given by Christopher Ham based on ideas drawn from a King's Fund Institute Briefing Paper (no. 6, 1988) on "Medical negligence:

compensation and accountability". In it, two other options for reform of the current system are discussed: modifying the existing tort system or abandoning it altogether in favour of providing compensation through social security.

There is a trenchant but moving paper given by Simanowitz who is critical of the "No fault" scheme on precisely those grounds: under such a scheme, the accountability of the medical profession would be reduced and the patients would lose out.

Space does not permit mention of all the papers or the issues raised in relation to them: separate schemes for drug injuries, neonatal/obstetric injuries, ways to improve accountability and the problem of funding such a scheme. As happens with books of this format, repetition of some viewpoints, and avoidance of others, does occur. Although in general, this is not a book for someone who knows nothing of the issues, there are some extremely good papers herein which would serve as an introduction. For the initiate, the paper by colleagues in the legal and insurance professions not only inform but also broaden the discussion in a stimulating way.

Finally, the meeting concluded that standards of care in medicine need to be improved and that the key issue is accountability. If one remembers that the focus of the discussion is really the benefit of injured patients, then this would seem to be correct.

GWEN ADSHEAD, *Senior Registrar in Forensic Psychiatry, St George's Hospital, Tooting*

Progress in Catecholamine Research. Part C: Clinical Aspects. Edited by R. H. BELMAKER, MERTON SANDLER and ANNICA DAHLSTROM. New York: Alan R. Liss. 1989. 540 pp. $165.00.

This is the last of a heavy triad of books devoted to catecholamines, in which all aspects of their function, from basic pharmacology through to clinical relevance in many medical disciplines, are examined. It represents the proceedings of a symposium held in Jerusalem in 1987 and is likely to be guaranteed sufficient sales from the many participants' institutions to pay for its publication.

Has it got enough interest for the rest of us, and for psychiatrists in particular? I think not. Despite over 300 pages devoted to catecholamines in affective disorders, schizophrenia, anxiety and aging, there is little to excite those grey cells orientated to clinical practice. The technology is tremendous, with barely a molecule of the main amines escaping detection in body fluids, but the relationship between neuropharmacology and clinical findings is so tenuous it appears to be as random as Brownian motion. Association does not equal cause, yet so often in these pages this assumption is made, so that everything from stress and bulimia to schizophrenic vulnerability is reduced to abnormalities in catecholamine function.

There are islands of excellence, particularly on imaging techniques in schizophrenia and some interesting speculations about the mechanism of antidepressant drug action, but these are not adequate compensation. Neither the psychiatrist nor the basic scientist will miss much by passing it by.

PETER TYRER, *Senior Lecturer, St Charles Hospital, London*

Trauma Transformation and Healing: an Integrative Approach to Theory, Research and Post-Traumatic Therapy. By JOHN P. WILSON. New York: Brunner/Mazel. 1989. 345 pp. $40.00.

Largely written by Wilson, this book is the fourteenth in the *Psychosocial Stress* book series. It comprises three sections, each of three chapters. The first section focuses on theoretical issues, including the influence of psychobiological factors, such as neurotransmitters, and cultural rituals and ceremonies on post-traumatic stress. In the second section, Wilson and his co-authors describe some of the empirical evidence confirming the complex ways that dispositional and environmental variables influence the individual's reactions to adversity. The empirical focus is principally on Wilson's own studies of Vietnam veterans and survivors of the Pearl Harbour Attack in 1941. Legal and treatment issues are discussed in the last section. Important principles of intervention are identified, including the need for prospective therapists to deal with countertransference (although some practitioners would question his use of this term) such as anger, disgust and guilt.

Two appendices (comprising 57 pages) contain copies of Wilson's main research instruments (the Vietnam Era Stress Inventory and the Pearl Harbour Personal Questionnaire). There are, in addition, accurately compiled name and subject indices and a reference section.

Exposure to trauma is not a new human experience and, although the inclusion of post-traumatic stress disorder (PTSD) in DSM–III catalysed much legal and clinical interest in the subject, we still have much to learn. In particular, efforts are required to integrate biological, social and psychological approaches, and for this reason Wilson's book is very welcome. It is not a completely comprehensive text because certain neurophysiological issues (e.g. hemispheric lateralisation) and certain developmental issues in traumatised children are not considered. Nonetheless, there is much in this book to fire the imagination and stimulate interest. In particular, Wilson gives a fascinating account of his own efforts to incorporate the features of an Indian healing ritual (the Sweat Lodge Ceremony) into a six-day intensive treatment programme for Vietnam veterans. Although there are methodological limitations to the study (e.g.

the sample is small and possibly not wholly representative of this group of survivors), the significant reduction in symptoms (especially of hostility) is worthy of further attention – as is the incidental finding that the wives were suspicious of and even ambivalent about the changes reported by their husbands.

In summary, Wilson and his co-authors have produced a useful book which emphasises the need for a holistic approach to PTSD. It is reassuring too that they emphasise the resilience of individuals in adversity and describe how some even derive positive gain therefrom.

DAVID A. ALEXANDER, *Senior Lecturer, Department of Mental Health, Medical School Buildings, Foresterhill, Aberdeen*

Tribunals on Trial. By JILL PEAY. Oxford: Clarendon Press. 1989. 256 pp. £27.50.

This book, written by an academic psychologist, is about mental health law, i.e. a set of rules and sanctions made by men and women in Parliament, and subject to change. It is also about justice – an attribution of right and wrong, by immutable criteria, to man in relation to other men and to society. This attempt to discharge two functions leads to difficulties and the use of strange epithets such as 'context-specific' justice, i.e. "Fiat justitia – if the circumstances are right".

Peay has been at pains to explore this dialectical position by first-hand observation of tribunals at work, and interviews with patients, their doctors, and members of tribunals.

Medicine, through the voices of the Responsible Medical Officer (RMO), and Law, through the judicial members of the tribunals, get a fair hearing, and it becomes obvious that neither discipline is averse to interpreting statutes selectively to further their respective aims of the best interests of the patient and of society. This seems to worry Peay, who is concerned that to the judges, "common sense was trumps" (p. 99).

As always (see Dell & Robertson's *Sentenced to Hospital*, 1988), the problems posed by the mentally ill are seen as relatively easy to solve; those of the psychopathically disordered, however, are the sources of most tortuous debate: for example, what should be done about the in-patient labelled as psychopathic but who is symptom-free in a milieu where heterosexual stimulation and alcoholic disinhibition are non-existent. Peay points out that "tribunals deal with the here and now, and psychiatrists with the past and future" (p. 78).

The Home Office is cast in the role of villain in the cases of involuntary patients. I learnt to my surprise that the Secretary of State has commissioned his own Independent Psychiatrist (IP) in some cases, and has been represented by counsel at hearings, in order to continue the detention of the patient. In one case, no fewer than seven IP reports were submitted (p. 120).

Another surprise to me was the contention that the medical member of the tribunal, a quasi-judicial body, should be subject to cross-examination by the patients' representative. Doctors will find many other statements and inferences in the book surprising. Those at 'Acheland' and 'Bendene' will not welcome the phrase "Once out of the clutches of the Special Hospital . . .".

Much space is devoted to the negative relationship between patients and the RMO which Peay says she found at Acheland. She seems unaware of the truism: "The work of custody creates a relation of opposition between those who exercise it and those upon whom it is exercised" (Smith, *Psychiatry*, **28**, 317).

Another surprise is the statement: "The tribunal were fully aware that in order for their recommendation (transfer from a locked to an open ward) to be put into effect, the Home Secretary would have to concur with this course". I can find no backing for this in the 1983 Act, the memorandum on the Act, or the Mental Health Review Tribunal rules.

In a pejorative context, Peay says of RMOs that "legal restrictions were simply not at the forefront of their minds". Why should they be? It is not only special hospital doctors who are assessed by rather odd criteria – at one tribunal the evidence of two IPs was preferred to that of the RMO since "both were regarded as reputable because they did not provide independent reports with any obvious regularity", i.e. the less you practice, the more likely you are to be perfect. Many more nits could be picked; perhaps trivial, but not conducive to ready acceptance of the burden of the arguments in the book. The author has an irritating habit of using a singular noun with a plural verb ("The Tribunal . . . *are* . . ."), some of her syntax is involved, and some sentences incomprehensible: "In the High Court these conflicting issues (i.e. patients rights at the expense of their care) are subsumed by the purity of the forum".

In the end, tribunals are seen to be a good thing, "a shop window for the public and a safety valve for the patient" (p. 69). Psychiatrists should read this book, at the cost of some mental dyspepsia; it will help them to wear their two hats to which Peay refers, perhaps improve their communications with the tribunals, and increase their 86% success rate in securing tribunal agreement to their views. However, for a book which will be consulted frequently by RMOs, IPs, and tribunal members, the index is disappointingly unhelpful.

PATRICK MCGRATH, *Physician Superintendent, Broadmoor Hospital, 1956–81, Medical Member, Mental Health Review Tribunal, 1960–84*

Contents of
THE AMERICAN JOURNAL OF PSYCHIATRY

Volume 147, Number 8 August 1990

Editorial

965 Utilization management managed or mangled psychiatric care? *Steven S Sharfstein*

Special Articles

967 Utilization management of mental health services by private third parties. *Gary L. Tischler*
974 The relation of ulcerative colitis to psychiatric factors: a review of findings and methods *Carol S. North, Ray E. Clouse, Edward L. Spitznagel and David H Alpers*

Regular Articles

982 Second-generation deinstitutionalization, I. the impact of *Brewster v. Dukakis* on state hospital case mix. *Jeffrey L Geller, William H. Fisher, Janet L Wirth-Cauchon and Lorna J. Simon*
988 Second-generation deinstitutionalization, II the impact of *Brewster v. Dukakis* on correlates of community and hospital utilization *Jeffrey L Geller, William H Fisher, Lorna J. Simon and Janet L Wirth-Cauchon*
994 Avoiding negligent release contemporary clinical and risk management strategies *Norman G. Poythress, Jr*
998 Drug use and life style among college undergraduates in 1989. a comparison with 1969 and 1978 *Harrison G. Pope, Jr, Martin Ionescu-Pioggia, Harlyn G Aizley and Deep K Varma*
1002 Substance use in borderline personality disorder *Rebecca A Dulit, Minna R Fyer, Gretchen L. Haas, Timothy Sullivan and Allen J Frances*
1008 Childhood sexual and physical abuse in adult patients with borderline personality disorder *Susan N. Ogata, Kenneth R. Silk, Sonya Goodrich, Naomi E. Lohr, Drew Westen and Elizabeth M Hill*
1014 Altered platelet α_2-adrenergic receptor binding sites in borderline personality disorders. *Steven M. Southwick, Rachel Yehuda, Earl L Giller, Jr and Bruce D. Perry*
1018 Bupropion treatment of attention-deficit hyperactivity disorder in adults. *Paul H. Wender and Fred W Reimherr*
1021 Diagnosis of major depression in cancer patients according to four sets of criteria *Roger G Kathol, Anand Mutgi, John Williams, Gerald Clamon and Russell Noyes, Jr*
1025 Syndrome comorbidity in patients with major depression or dysthymia· prevalence and temporal relationships. *William C Sanderson, Aaron T. Beck and Judith Beck*
1029 Evidence for reduced recovery from depression in patients taking benzodiazepines during unilateral ECT *Helen M. Pettinati, Stephani M. Stephens, Kenneth M Willis and Sarah E. Robin*
1036 The role of depression in couples involved in murder-suicide and homicide *Milton Rosenbaum*
1040 A comparison of manual and MEDLARS reviews of the literature on consultation-liaison psychiatry *Joseph C. Bareta, David B. Larson, John S. Lyons and Joseph J. Zorc*
1043 Validity of the personality diagnostic questionnaire – revised· comparison with two structured interviews. *Steven E. Hyler, Andrew E Skodol, H David Kellman, John M Oldham and Lyle Rosnick*
1049 Psychiatric symptoms and nursing home placement of patients with Alzheimer's disease. *Cynthia Steele, Barry Rovner, Gary A Chase and Marshal Folstein*
1052 Prediction of adult-onset schizophrenia from childhood home movies of the patients *Elaine Walker and Richard J. Lewine*
1057 Failure to detect fabricated posttraumatic stress disorder with the use of the MMPI in a clinical population *Stephen T. Perconte and Anthony J Goreczny*
1061 Developmental history and object relations in psychiatrically disturbed adolescent girls. *Drew Westen, Pamela Ludolph, M. Judith Block, Jean Wixom and F. Charles Wiss*

Clinical and research reports

1069 Clozapine and seizures. *Ellen Haller and Renee L Binder*
1072 Growth hormone response to growth hormone-releasing hormone in schizophrenic patients. *David I Mayerhoff, Jeffrey A. Lieberman, Carmen Z Lemus, Simcha Pollack and Bruce S. Schneider*
1075 Role of cigarette use in hyponatremia in schizophrenic patients *Michael Allon, Henry M. Allen, Larry V. Deck and Mervin L. Clark*

Contents of The Journal of Nervous and Mental Disease

August 1990 **Volume 178, Number 8**

Epidemiological analysis of alcohol and drug use as risk factors for psychotic experiences. *Allan Y. Tien and James C. Anthony* 473

Thought disorder in the relatives of schizophrenics. A meta-analytic review of selected published studies. *David M. Romney* 481

Linguistic and cognitive failures in schizophrenia. A multivariate analysis. *Philip D. Harvey and Mark R. Serper* 487

Cognitive functioning and treatment outcome in alcoholics. *Arthur I. Alterman, Harvey Kushner and John M. Holahan* 494

The lifetime prevalence of psychopathology in men with multigenerational family histories of alcoholism. *Peter R. Finn, Irwin Kleinman and Robert O. Pihl* 500

Adult children of alcoholics. A valid diagnostic group? *Ann Ivy Fulton and William R. Yates* 505

The positive and negative syndrome scale – Spanish adaptation. *Stanley R. Kay, Abraham Fiszbein, Marc Vital-Herne and Luis Silva Fuentes* 510

Predicting outcome in schizoaffective psychosis. *Thomas H. McGlashan and Paul V. Williams* 518

Dementia in schizophrenia. Magnetic resonance and clinical correlates. *Alessandro Rossi, Silvana Galderisi, Vittorio Di Michele, Paolo Stratta, Simona Ceccoli, Mario Maj and Massimo Casacchia* 521

Avoidant coping predicts psychological disturbance in the elderly. *Lawrence W. Smith, Thomas L. Patterson and Igor Grant* 525

Anorexia nervosa in 19th century America. *Walter Vandereycken and Eugene L. Lowenkopf* 531

Brief Reports

Characteristics of psychiatric inpatients who assault staff severely 536

Recurrence of puerperal psychosis not prevented by prophylactic progesterone administration 537

Body dysmorphic disorder: the relationship to obsessive-compulsive disorder 538

Book Reviews. Bytheway, B., Keil, T., Allatt, P. and Bryman, A. (eds) *Becoming and Being Old. Sociological Approaches to Later Life;* Cooper, A. M., Kernberg, O. F. and Person, E. S. *Psychoanalysis: Toward the Second Century;* Elizur, J. and Minuchin, S. *Institutionalizing Madness. Families, Therapy and Society;* Brody, H. *Stories of Sickness;* Oren, N. and Abramsky, O. *Neurosciences in Israel;* Dell, S. and Robertson, G. *Sentenced to Hospital: Offenders in Broadmoor;* Dowie, J. and Elstein, A. (eds) *Professional Judgment: A Reader in Clinical Decision Making;* Greist, J. H., Carroll, J. A., Erdman, H. P., Klein, M. H. and Wurster, C. R. (eds) *Research in Mental Health Computing: The Next Five Years;* Kren, G. M. and Christakes, G. *Scholars and Personal Computers: Microcomputing in the Humanities and Social Sciences;* Rosen, R. C. and Beck, J. G. *Patterns of Sexual Arousal: Psychophysiological Processes and Clinical Applications* 541

Index to Advertisers

Pharmaceuticals

The Boots Company plc	*facing* p 326
Duphar Laboratories	*facing* p 327
Janssen Pharmaceuticals Ltd.	*facing* p 331
Janssen Pharmaceuticals Ltd.	*facing* p 338
Janssen Pharmaceuticals Ltd.	*facing* p 339
Lederle Laboratories Ltd.	*facing* p 326
Lederle Laboratories Ltd.	*facing* p 330
Lundbeck Limited	inside front cover
Lundbeck Limited	outside back cover
E. Merck Ltd.	*facing* p 330
E. R. Squibb	p x

Appointments

Assoc. of Commonwealth Universities	p vi
Government of Nova Scotia	p iv
Medicine Hat Regional Hospital	p iv
Medicine Hat Regional Hospital	p viii

Booksellers & Publishers

Springer-Verlag	p v
John Wiley & Sons Ltd.	p vii

Clinics, Hospitals & Nursing Homes

St. Andrew's Hospital	p ix

Miscellaneous

Munro Clinic	p viii
Psychiatrische Universitatsklinik	p viii
Southmead Hospital	p iv

Printed in Great Britain by Henry Ling Ltd., at the Dorset Press, Dorchester, Dorset

STORMS BLOW
FIND THE EYE
WINDS CHANGE
... REFLECT (Anon)

CLOPIXOL®
NEW ACUPHASE
zuclopenthixol acetate ▼

A harmonious new way to treat acute psychosis

Lundbeck

FACING THE FUTURE TOGETHER

Further information is available on request from:

1990

30. 4. 91

THE BRITISH JOURNAL OF PSYCHIATRY

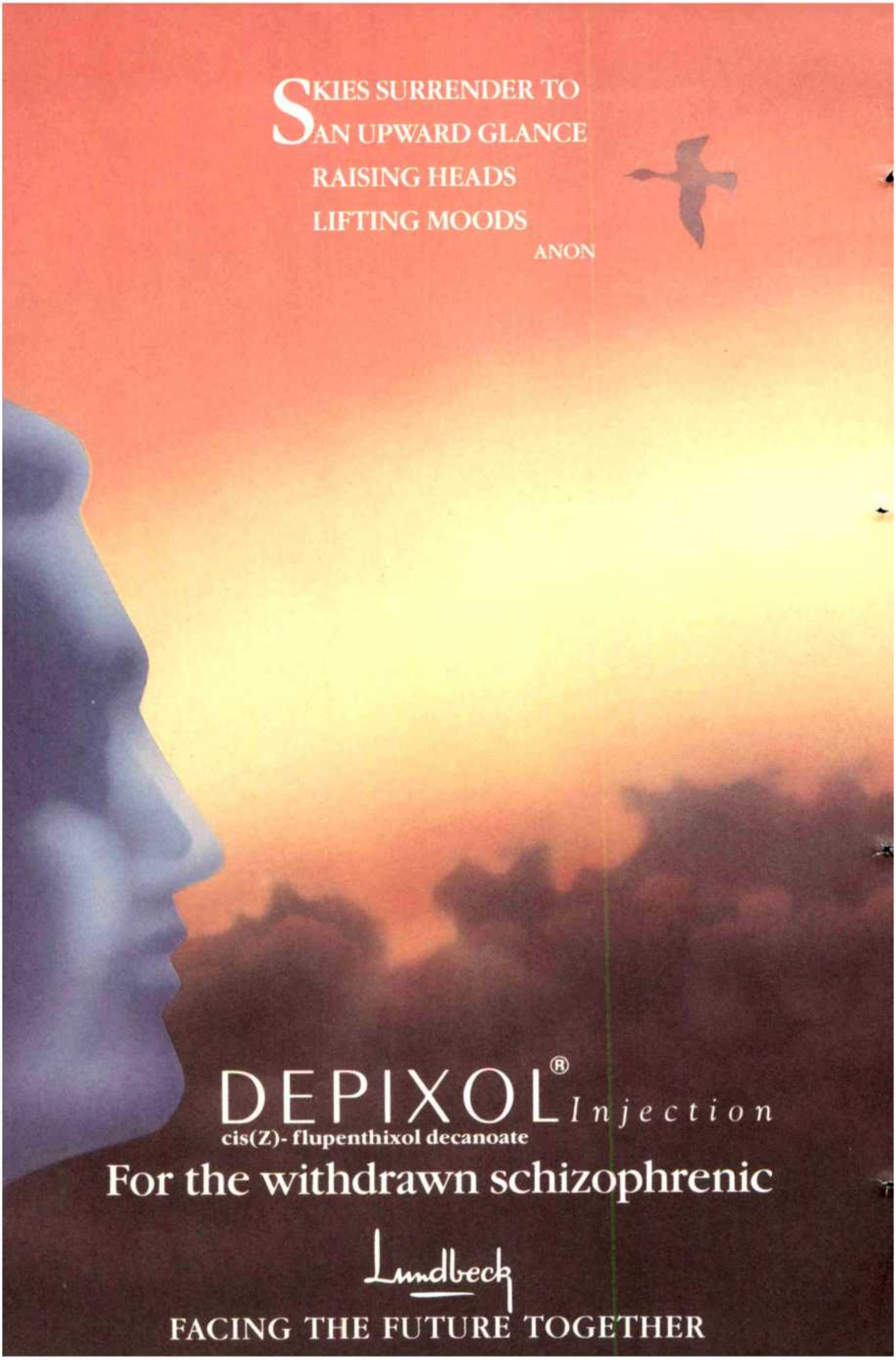
October 1990 Vol 157

UCM

Bowker

Published by
The Royal College of Psychiatrists

ISSN 0007-1250



SKIES SURRENDER TO
AN UPWARD GLANCE
RAISING HEADS
LIFTING MOODS
ANON

DEPIXOL® *Injection*
cis(Z)- flupenthixol decanoate

For the withdrawn schizophrenic

Lundbeck

FACING THE FUTURE TOGETHER

THE BRITISH JOURNAL OF PSYCHIATRY

October 1990 Volume 157

Annotation

Psychological sequelae of torture. A descriptive model. *S. Turner and C. Gorst-Unsworth* 475

Lecture

Structure and function in neurology and psychiatry. *E. H. Reynolds* 481

Review Article

Overexcitement and disinhibition. Dynamic neurotransmitter interactions in alcohol withdrawal. *P Glue and D Nutt* 491

Papers

Towards a system of outcome indicators for mental health care. *R. Jenkins* 500

The functional psychoses in Afro-Caribbeans. *I. Harvey, M. Williams, P. McGuffin and B. K. Toone* 515

Eating behaviour and attitudes to weight and shape in British women from three ethnic groups. *B. Dolan, J. Hubert Lacey and C. Evans* 523

Suicide in England and Wales and in Scotland. An examination of divergent trends *I. K. Crombie* 529

Suicide in Cork and Ireland. *M. J. Kelleher and M. Daly* 533

Adult affective disorder and early environment. *B. Rodgers* 539

Hormonal responses to fenfluramine in depressive subtypes. *P Mitchell, G. Smythe, G. Parker, K. Wilhelm, I. Hickie, H. Brodaty and P Boyce* 551

Syndromes of chronic schizophrenia. *P. F. Liddle and T. R. E. Barnes* 558

Regional brain metabolism during auditory hallucinations in chronic schizophrenia. *J. M. Cleghorn, E. S. Garnett, C Nahmias, G. M. Brown, R. D. Kaplan, H. Szechtman, B. Szechtman, S. Franco, S. W Dermer and P. Cook* 562

A trial of family therapy versus a relatives' group for schizophrenia. Two-year follow-up. *J. Leff, R. Berkowitz, N. Shavit, A. Strachan, I. Glass and C. Vaughn* 571

Obstetric complications, the putative familial-sporadic distinction, and tardive dyskinesia in schizophrenia *E. O'Callaghan, C. Larkin, A. Kinsella and J. L. Waddington* 578

Predictors of improvement in tardive dyskinesia following discontinuation of neuroleptic medication. *W. M Glazer, H Morgenstern, N. Schooler, C S. Berkman and D. C. Moore* 585

Symptom specificity in patients with panic. *P. Lelliott and C. Bass* 593

Brief Reports

Prevalence of Huntington's disease among UK immigrants from the Indian subcontinent. *R. S. Shiwach and R. H. Lindenbaum* 598

Can brain damage protect against schizophrenia? A case report of twins. *S. W Lewis, I. Harvey, M. Ron, R. Murray and A. Reveley* 600

Claimed simulation of insanity. A coping strategy in mania. *T. R. Suresh and T. N. Srinivasan* 603

Reflections of the self: atypical misidentification and delusional syndromes in two patients with Alzheimer's disease *S E. Molchan, R. A Martinez, B A. Lawlor, J. H. Grafman and T. Sunderland* 605

Delusional misidentification, AIDS and the right hemisphere. *P. Crichton and S. Lewis* 608

Rapid-cycling disorder in association with cerebral sarcoidosis *D. G. Walbridge* 611

Temporary Munchausen syndrome. *P M. Higgins* 613

Correspondence 617

A Hundred Years Ago. *Researched by Henry Rollin* 626

Books Reconsidered

Bereavement. Studies of Grief in Adult Life (C. M. Parkes) *David A. Alexander* 627

Book Reviews 630

American Journal of Psychiatry, Volume 147, Number 9, September 1990 644

The Journal of Nervous and Mental Disease, Volume 178, Number 9, September 1990 *facing* 644

CAES CENTRAL LIBRARY

St. Andrew's at Harrow

Bowden House Clinic

Relaunched after total refurbishment, the clinic offers treatment for:

Acute psychiatric illness
Stress/Anxiety problems
Alcohol problems
Eating Disorders
Elderly mentally infirm
Marital problems

The Clinic is set in a wooded oasis five minutes walk from Harrow School and shops. A peaceful sanctuary so near to Central London.

The aim of **St. Andrew's** is to provide the highest quality psychiatric diagnostic assessment, treatment, rehabilitation and continuing care facilities in a pleasant, caring and friendly environment.

Specialists in Psychiatric Medicine for over 150 years St. Andrew's Hospital is an independent 'not for profit' charitable trust.

For further details write or telephone:
Consultant-In-Charge

St. Andrew's at Harrow

Bowden House Clinic
London Road
Harrow-on-the-Hill
Middlesex HA1 3JL
Telephone: 081 864 0221
Fax: 081 864 6092

The British Journal of Psychiatry

October 1990, Volume 157

Published monthly. Founded in 1853 as the *Asylum Journal* and known as the *Journal of Mental Science* from 1855 to 1963

Editor: Hugh Freeman

Senior Associate Editor
Alan Kerr

Associate Editors
Dora Black, Sidney Crown, Julian Leff, Sir Martin Roth, Peter Tyrer

Assistant Editors
Mohammed Abou-Saleh, Peter Bruggen, Patricia Casey, Alexander Kellam, Stuart Montgomery, Catherine Oppenheimer, Ian Pullen, Rosalind Ramsay, Henry Rollin, Oliver Russell, George Stein, Digby Tantam, Greg Wilkinson, Stephen Wilson

Publications Manager
Dave Jago

Scientific Editor
Ralph Footring

Bulletin Manager
Elaine Millen

Assistant Scientific Editor
Katy Wimhurst

Editorial Assistants
Judy Ashworth, Ian O'Reilly, Christine Connolly

⊛™ The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences—Permanence of Paper for Printed Library Materials. ANSI Z39. 48–1984.

Printed by Henry Ling Ltd, The Dorset Press,
23 High East Street, Dorchester, Dorset DT1 1HD
(tel. 0305-251 066).

©1990 The Royal College of Psychiatrists

Subscriptions
Non-members of the College should contact the British Journal of Psychiatry Subscription Department, The Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. Annual subscription rates for 1990 (12 issues post free) are as follows:

UK: institutions £105, individuals £86
US: institutions $265, individuals $162
Elsewhere: institutions £132, individuals £106
Single copies of the Journal, £10, $20 (post free)

Payment should be made out to the British Journal of Psychiatry.

Queries from non-members about missing or faulty copies should be addressed within six months to the same address; similar queries from College members should be addressed to the Registration Department, The Royal College of Psychiatrists, 17 Belgrave Square, London SW1X 8PG.

Advertising
Correspondence and copy should be addressed to Peter T. Mell, Advertisement Manager, PTM Publishers Ltd, 282 High Street, Sutton, Surrey SM1 1PQ (tel. 081-642 0162, fax 081-643 2275).

US Mailing Agents: Mercury Airfreight International Ltd. Inc., 2323 Randolph Avenue, Avenel, NJ 07001.

Back issues published before 1988 may be purchased from William Dawson & Sons Ltd, Cannon House, Folkestone, Kent, England (tel. 0303-850-101).

STAFF PSYCHIATRIST

Nova Scotia, Canada

Applications are invited for the position of a Staff Psychiatrist to join our complement of 6.4 Psychiatrists at Yarmouth Regional Hospital. Yarmouth is situated in Southwestern Nova Scotia. It is well connected by road, air and sea to the provincial capital of Halifax as well as other major Atlantic Canadian and US cities. The town affords good shopping, inexpensive housing and other amenities.

Psychiatric mental health services provided by the fully accredited 183 bed Yarmouth Regional Hospital include a 20 bed acute Psychiatric inpatient unit, a day hospital, a well staffed outpatient department, and three satellite clinics which are run in neighbouring towns.

We are looking for a general Psychiatrist with leadership qualities who is a member of the Royal College of Psychiatrists. Candidates who have passed the Medical Council of Canada Evaluating Examination (MCCEE) will be preferred, but others are also encouraged to apply.

The recent introduction of remuneration through a contract arrangement makes this a very attractive position as it significantly enhances the potential salary – $103,000 to $130,000 on a 37.5 hour work week and may be augmented with extra reimbursement for call back. In addition extra billing may be available where staff complement permits and private practice is possible. The option also continues to exist for applicants who prefer a salary arrangement with a full range of benefits rather than a contract basis.

Assistance with moving expenses and bonuses totalling up to $25,000, dependent upon qualifications, are also available. In accordance with current income tax interpretation the bonuses are non-taxable.

Please send a Curriculum Vitae to or for further information contact:

Director of Human Resources
Yarmouth Regional Hospital
60 Vancouver Street
Yarmouth, Nova Scotia, B51 2P5
Canada
Telephone: (902) 724-3541, Ext. 251.

PSYCHIATRIST

LANGLEY, BRITISH COLUMBIA CANADA

Langley Memorial Hospital, a 200 bed general hospital, requires an additional 3 psychiatrists to fill positions in our Psychiatric Department. The successful applicants would join a group of 2 consultants currently on staff at this hospital. Remuneration is via Medical Services Plan fee-for-service. There is also ample opportunity for private office practice as well as sessional work with adjacent Central Health Care Centre. Langley, in the Lower Fraser Valley, has a community population of 76,000, and is a rapidly growing area, 45 minutes from downtown Vancouver. It thus produces the tranquility of small-town rural life with easy access to a sophisticated metropolis. Candidates must be prepared to sit Canadian Qualifying Exams. Submit curriculum vitae with the names of three references to:

Dr L. J. Rostvig
Vice-President Medicine
Langley Memorial Hospital
22051 Fraser Highway
Langley, British Columbia
Canada V3A 4H4

Dr Rostvig will be conducting interviews in London during November 1990.

British Journal of Psychiatry (1990), **157**, 475–480

Annotation

Psychological Sequelae of Torture
A Descriptive Model

STUART TURNER and CAROLINE GORST-UNSWORTH

Torture is one of the most important preventable causes of psychological morbidity. Amnesty International (1987) has reported the use of "brutal torture and ill-treatment" in over 90 countries in the 1980s. In some countries torture has been applied on such a widespread scale and in such an arbitrary manner that whole populations are affected. In Kampuchea under the Pol Pot regime, for example, genocide and torture took place on a massive scale; indeed, merely wearing spectacles became for many a capital offence (Amnesty International, 1983). Similar reports abound from many other countries and regions.

Torture is hard to define satisfactorily but some of the important elements may be found in the United Nations (1984) convention against torture. It is an act by which severe pain or suffering (physical or psychological) is intentionally inflicted on a person for such purposes as: (a) obtaining information; (b) obtaining a confession; (c) punishment; (d) intimidation or coercion; (e) any reason based on discrimination. The common theme is the desire to accomplish the purpose of the torturer against the will of the victim. The UN convention also makes it explicit that torture is always carried out by, or with the agreement of, a public official. Torture defined in this way is essentially the act of a state against an individual or group, with the aim of achieving specific psychological changes (directly) in their victims and often (indirectly) in their communities.

Although a great deal has been written about the history and the methods of torture, and moving testimonies have been produced by survivors, there is still no adequate framework within which to describe the range of psychological reactions reported.

There have been attempts to describe a single 'torture syndrome' (Allodi & Cowgill, 1982; Abildgaard *et al*, 1984; Basoglu & Marks, 1988) but these are generally unconvincing. They lack a theoretical base and appear to be no more than a list of common symptoms seen in survivors of torture. These include impaired memory and concentration, headache, anxiety, depression, sleeplessness with nightmares and other intrusive phenomena, emotional numbing, sexual disturbances, rage, social withdrawal, lack of energy, apathy, and helplessness (Cathcart *et al*, 1979; Rasmussen & Lunde, 1980; Allodi & Cowgill, 1982; Abildaard *et al*, 1984). In a review, Goldfeld *et al* (1988) attempted to introduce some order by breaking down these symptoms into three categories: cognitive, psychological, and neurovegetative. This may be useful as a descriptive aid but it lacks a theoretical (aetiological) basis. The range of symptoms listed is very wide and includes features of many of the common psychiatric disorders.

Some would argue that it is invalid to make any attempt to produce a nosology of torture reactions. For example, it has been suggested that torture is a political process and its sequelae should be understood primarily in political terms (Martin-Baro, 1988). To take a contrasting case, early psychoanalytic theories emphasised fantasy rather than actual memories as being all important in the development of psychological disturbance. Seen in this way, an individual's reaction to torture could only be understood in the light of that person's own unique set of formative experiences. However, since Freud's work with the war neuroses, psychoanalytic thinking now recognises that the pre-existing personality and the vulnerability of the ego are only part of the multifactorial causation of a particular individual's reaction to trauma (Niederland, 1968; Krystal & Niederland, 1971; Moses, 1978; Bergmann & Jucovy, 1982). We would argue that many of the reactions to torture should be understood in psychological as well as political terms and that descriptions of common syndromes can be useful both theoretically and therapeutically.

We acknowledge that individuals react differently to torture, as they do to many other psychological stressors. Although the primary aim of torture is to effect a specific psychological change, this will vary for different people in different situations. Subjugation of the will of the victim to that of the torturer is the common theme. However, for some it will be one aspect of interrogation, for others it will be punishment, and for many it will form part of the process of political, tribal or religious repression. It is partly for this reason that we reject the approach which relies on the use of a single 'torture syndrome'.

It is our contention that torture is an activity with particularly complex psychological and physical sequelae. The survivor of torture has not merely been the accidental victim of physical injury or threat of death such as might occur, for example, in a natural disaster or accident (and these are disturbing enough). He or she has received the focused attention of an adversary determined to cause the maximal psychological change. This perversion of an intimate relationship (Ritterman, 1987) for sociopolitical reasons will often include the deliberate intention to disrupt the normal healing process and thus maximise the psychological scars.

Neither is it just the individual who suffers. For every person tortured there are mothers and fathers, wives, husbands and children, friends and relatives who wait in uncertainty and fear (Cohn *et al*, 1985; Ritterman, 1987). Torture has effects on communities and on whole societies. This dimension must be included in any effective model.

Developments in the social and clinical sciences during this century have provided the opportunity for advancement in our understanding of the long-term reactions to torture. In this paper, a framework is presented which we believe might form the basis for a nosology of reactions to torture. It has been developed from our substantial clinical experience obtained with a London-based group, the Medical Foundation for the Care of Victims of Torture (Turner, 1989). Not only are these dimensions of heuristic value, they also stand to have important implications for treatment.

Common elements of the response to torture

(a) Individuals

(i) Incomplete emotional and cognitive processing

The central features of most specific stress-response syndromes are (a) the compulsive tendency towards repetition of some aspect of the disturbing experience (as thoughts of the original event, as feelings associated with the experience, or as behavioural re-enactments), (b) associated hyperarousal, and (c) the defensive avoidance of internal or external representations which recall or resemble the traumatic episode (Horowitz, 1976). These have come to assume central importance in post-traumatic stress disorder (PTSD), outlined in DSM-III-R (American Psychiatric Association, 1987).

Several explanatory models for this process have been put forward, including the repetition-compulsion principle of Freud (Horowitz, 1976), traumatic visual imagery (Brett & Ostroff, 1985), and the effects of conditioning (Wirtz & Harrell, 1987; Basoglu & Marks, 1988). However, the most convincing explanation rests on a model of emotional processing. Horowitz suggests that painful experiences need to be processed in the mind, in the same way as any other formative experience. In this view he is supported by Rachman (1980), who writes of emotional processing as a normal way of dealing with affective material. If this processing is arrested or incomplete, it is then a source of continuing distress for the individual. Horowitz suggests that there are natural (protective) limits to the rate of cognitive and emotional processing. Material which threatens to overwhelm the person leads to the invocation of these defensive mechanisms involving "massive ideational denial and emotional numbing". In his theoretical and descriptive work, he points to a cyclical process in which the individual shifts between avoidance with denial and a state of emotional distress associated with intrusive memories and thoughts. In extreme cases, a patchy amnesia for the traumatic event may be induced. To move on from this condition, and complete the task of processing, requires the intrusion of manageable chunks ('doses') of this material into consciousness. In this way, successful processing is accompanied by a phasic process of intrusion alternating with denial. Of course, the traumatic event may be brought into consciousness not just by internal systems but also by external stimuli which resemble the circumstances of the traumatic episode; there may be frank avoidance of such stimuli.

In other words, the person is seen as having created internal (cognitive) schemata of the external world against which external information is measured. Events which are incongruent with this world picture lead to changes in the internal representation in the person's mind through cognitive and emotional processing. Massive stressors have the effect of overwhelming this normal process and morbid reactions result.

These intrusive, hyperarousal and avoidance phenomena undoubtedly occur in people who have been subjected to torture (Mollica *et al*, 1987). Indeed in torture, where the infliction of physical and psychological pain can continue over many months, the reactions may be particularly severe. The deliberate application of torture at a time which suits the torturer and which is intended to have the maximum impact on the victim makes it much more difficult for the victim to mount any satisfactory defence. It may be predicted that survivors of torture are less able to control their later intrusive recollections than victims of other forms of trauma of similar intensity.

Factors that stand to impede emotional processing have been described (Rachman, 1980); several of these seem to be commonplace in torture. For example, high arousal, fatigue, sleeplessness and the irregularity of presentation of dangerous, intense, uncontrollable traumas are all said to militate against successful processing. Some survivors have learned (often by chance) methods of reducing the pain of torture. Such operant behaviour may include head banging as a way of losing consciousness or hyperventilation as a way of dissociating from the impact of the violence. Following torture, other methods of avoidance may develop; these include phobic postures and marked changes in behaviour.

The impact of prolonged exposure to painful stimuli, and the ever-present threat of a recurrence of attack, may lead to a state of marked hyperarousal and chronic anxiety during detention. Following release, survivors are often vulnerable to repeated arrests; even if they escape the country where torture took place they may equally be subject to detention and forcible repatriation. They are likely to deal with these chronic anxiety symptoms by using the responses learned during torture. Of course these may now be even more maladaptive.

(ii) Depressive reactions and life events

Surrounding the experience of torture are many emotionally charged processes chiefly concerned with loss. Individual survivors of torture may have lost parts of their bodies (limbs or eyes for example), normal bodily functions, or bodily health. They are likely to have lost work, status, family, and credibility. Even if they succeeded in resisting torture their colleagues are likely to be suspicious of them. If they stay in the same region they know that there is the continuing threat of repeated detention and torture. They know that there are similar threats to family and friends. If they leave the region to seek asylum elsewhere these losses are compounded. Torture therefore must be seen not only as a very important life event in its own right but also as the cause of many others.

A depressive reaction is a common sequel to torture and is almost certainly related to these loss events (Brown & Harris, 1978). This is particularly likely to be the case when the individual is examined as an asylum seeker or refugee (Kinzie *et al*, 1984). In a recent American series of 52 South-East Asian refugees (Mollica *et al*, 1987) only one subject had PTSD alone; the remaining 25 individuals with PTSD also had another diagnosis, usually major affective disorder. Indeed, major depression was the most common diagnosis in the whole sample, being diagnosed in 37 of the 52 subjects. Those Kampuchean widows who had experienced at least two of the three traumas of rape, loss of spouse or loss of children had the highest levels of depressive symptoms. They perceived themselves as socially isolated and living in a hostile social world.

(iii) Somatic symptoms

Symptoms suggestive of possible organic disease appear to constitute another dimension of the torture reaction (Cathcart *et al*, 1979; Rasmussen & Lunde, 1980; Rasmussen & Marcussen, 1982). These may be the direct consequences of torture (Gordon & Mant, 1984; Goldfeld *et al*, 1988) although in people whose bodies have been tortured as a way of gaining control over their minds, somatic symptoms may have a multitude of meanings. Not infrequently they appear to be intimately related to a disturbed emotional state; sometimes the chronic hyperventilation syndrome (Pincus, 1978) appears to be an important mechanism.

The aetiology of cognitive impairment following torture is similarly unknown. It is likely to be a consequence of the two factors of disturbed emotional state leading to a 'pseudodementia' (Lishman, 1987) and recurrent brain damage (Goldfeld *et al*, 1988). Common injuries which may contribute to organic damage include not only physical blows to the head, but also anoxic episodes in *submarino* torture, deliberate deprivation of food leading to malnutrition, the effects of chronic illness, and the use of recurrent electrical shocks. Sailors in wartime who had been provided with good physical circumstances but who had endured prolonged periods of danger have also been reported to show frequent memory impairment and reduced concentration (Askevold, 1980). This suggests that psychological factors are sufficient to account for cognitive impairment. However, in torture survivors it is impossible to rule out significant physical damage to the brain as an additional factor.

Finally, sexual dysfunction is often reported, although the mechanism of this is also unclear (Lunde *et al*, 1981). It is likely that psychological factors are very important. Sexual violence is a common form of torture especially in women and female adolescents (Goldfeld *et al*, 1988).

(iv) The existential dilemma

We have given the title 'existential dilemma' to the effect of torture which is perhaps the most difficult to conceptualise in medical terms, but may be the most enduring and important aspect of torture. With torture, as with some other forms of extreme trauma, it is important to consider the broader personal, social, and political dimensions. People are tortured for a purpose. Often it is not that the individual has important information or that a confession is required: if a regime is acting outside the usual rules of law there are simpler ways of investigation, and people may be punished or killed ('disappeared') without proof being required. It seems that torture is used for a combination of reasons, but chief among these are punishment and systematic repression of whole communities. Membership of a political, social, tribal or religious group may be sufficient reason in itself for a person to be detained, tortured and killed (Stover & Nightingale, 1985).

Torture is a "catastrophic existential event" (Bendfeldt-Zachrisson, 1985). Survivors of torture face the double dilemma both of coming to terms with the full reality of torture in their spiritual and political understanding of their world and also of surviving unchanged the insidious pressure of the torturer to change – to act or react in relation to his wishes. The mere act of survival may bring its own guilt, a phenomenon which has been described in reactions to other forms of traumatic stress (e.g. Horowitz, 1976). Sometimes survivors of torture also have to remember other more shameful actions they had to perform to secure their own lives.

The need to live in a world of such unremitting cruelty is the material of testimony and autobiography (e.g. Levi, 1987). The purpose of existence itself is challenged by the fact of torture. Perhaps it is in this area that early experiences and political or religious culture are likely to have the strongest effects. They stand to influence the individual's vulnerability to torture. It is also possible that those survivors who come from active political or religious groups and who are able to return to their struggle may obtain sufficient peer support to help them overcome some of these broader effects of torture on their internal lives. Those who are broken by torture and who have to escape for safety may find themselves in a joyless world in which they can find little personal meaning or fulfilment. They must to some extent come to reconcile the 'new self' with the 'new reality' of the external world.

One of the Medical Foundation's patients has described the members of a self-help group he attended in this way: "This is a chain, one link and then another. It will be a great chain. When we eventually shake this chain it will be like thunder. You will hear thunder. The world will stop to listen, humanity will come to its senses and there will be no more torture. . . ." (Schlapobersky & Bamber, 1988). In making this assertion, he is trying to reclaim for himself a personal meaning, to find his way out of the existential despair he had experienced and to stake his claims on the future he would like to see.

(b) Families and communities

The broader psychological effects in the survivor's family and community are also important. Particularly in close relatives, the problems in each of the three dimensions of traumatic stress reaction, depression and the existential dilemma, may be as troublesome as in the survivors themselves.

Children of survivors of torture have been investigated and show evidence of psychological disturbance (Allodi, 1980; Cohn *et al*, 1980, 1985). All who come so close to torture will have to come to terms with a world in which barbaric violence is not only theoretically possible, but is also a stark reality in their own lives. For children particularly, this may come to have profound and lasting effects.

Torture and killing of individuals may have a striking effect on the social and political life of a country or region (Argentina's National Commission on Disappeared People, 1986). Although outside the main area of this paper, it is a dimension which cannot be ignored in developing services to assist in the rehabilitation of survivors (Martin-Baro, 1988).

Discussion

The multidimensional approach has at least three significant advantages over the use of a single torture syndrome. It moves away from a narrow medical perspective and underlines the complexity of torture and its sequelae. It is based on aetiological models which may be subjected to scientific investigation. Finally, it introduces some of the common attitudinal, social, and political consequences of torture.

The introduction of clinical psychiatric diagnoses in this field has been of enormous value to victims of traumatic stress. Thus concentration-camp survivors have been compensated for psychological symptoms, and both US Vietnam war veterans and UK disaster survivors have been eligible for more energetic and easily accessible psychological assistance. However, not all reactions can or should be reduced to the level of categorical medical diagnoses. Particularly in relation to state-sanctioned torture, it is important to acknowledge that there are significant social and political sequelae and that although these may be reflected in attitude changes in individual survivors, they are actually a part of a larger process.

For each individual the pattern of different reactions is likely to guide the approach to treatment; it also makes the limitations of treatment *per se* more explicit. For example, if the intrusive phenomena are dominant, some would wish to prescribe an antidepressant drug (Boehnlein *et al*, 1985) or to consider a process of exposure and habituation

(Basoglu & Marks, 1988). If the depressive symptoms are predominant then antidepressant drugs or alternatively a psychological treatment for depression might be the first treatment of choice. However, if the social, political and existential changes dominate, then the longer and more difficult process of healing and readaptation is required. This work is likely to depend on many different agencies. For some the political group has prime importance; for others it may be the religious leader; for a proportion it will fall to the health profession to offer help. Some therapists have found the formal use of a technique based on the writing of a testimony to be an important part of this work (Cienfuegos & Monelli, 1983). This not only encourages the individual to recall and work through (emotionally process) the trauma story, it also helps to set the torture experience in perspective. It allows individuals to gain a more objective appraisal of their own lack of freedom under torture. In a deliberate way, people may have been made to feel somehow responsible for their own torture. A technique like testimony encourages a restructuring of these cognitions and helps people to understand their personal dilemma within a larger political context.

As has already been acknowledged, survivors may find strength and recovery by re-engaging in their former struggle. For asylum seekers and refugees in strange countries, the knowledge that others are in the same position and are struggling to reach their own understandings may be very powerful; the group-therapeutic approach over substantial periods of time may be the best way of helping such people to deal with these problems. These groups combine self-help principles and psychotherapeutic insights in a creative way, helping individuals and groups to find their own solutions, to reassert their own rights to personal existence (Schlapobersky & Bamber, 1988). For those survivors fortunate enough to be in a safe country with their partner or family, it is probably important to offer them therapy as a couple or family (Svendsen, 1985). It is our experience that a flexible, patient-centred approach using several techniques in combination or at different stages of recovery achieves the best results.

References

Abildgaard, U., Daugaard, G., Marcussen, H. *et al* (1984) Chronic organic psycho-syndrome in Greek torture victims. *Danish Medical Bulletin*, 31, 239–242.

Allodi, F. (1980) The psychiatric effects in children and families of victims of political persecution and torture. *Danish Medical Bulletin*, 27, 229–232.

—— & Cowgill, G. (1982) Ethical and psychiatric aspects of torture. *Canadian Journal of Psychiatry*, 27, 98–102.

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders (3rd edn, revised) (DSM-III-R)*. Washington, DC: APA.

Amnesty International (1983) *Political Killings by Governments*. London: Amnesty International Publications.

—— (1987) *Amnesty International Report 1987*. London. Amnesty International Publications.

Argentina's National Commission on Disappeared People (1986) *Nunca Mas (Never Again)*. London: Faber & Faber.

Askevold F. (1980) The war sailor syndrome. *Danish Medical Bulletin*, 27, 220–223.

Basoglu, M. & Marks, I. (1988) Torture; research needed into how to help those who have been tortured. *British Medical Journal*, 297, 1423–1424.

Bendfeldt—Zachrisson, F. (1985) State (political) torture: some general, psychological and particular aspects. *International Journal of Health Services*, 15, 339–349.

Bergmann, M. S. & Jucovy, M. E. (eds) (1982) *Generations of the Holocaust*. New York: Basic Books.

Boehnlein, J. K., Kinzie, J. D., Ben, R. *et al* (1985) One-year follow-up study of posttraumatic stress disorder among survivors of Cambodian concentration camps. *American Journal of Psychiatry*, 142, 956–959.

Brett, E. A. & Ostroff, R. (1985) Imagery and posttraumatic stress disorder: an overview. *American Journal of Psychiatry*, 142, 417–424.

Brown, G. W. & Harris, T. (1978) *Social Origins of Depression*. London: Tavistock.

Cathcart L. M., Berger, P. & Knazan, B. (1979) Medical examination of torture victims applying for refugee status. *Canadian Medical Association Journal*, 121, 179–184.

Cienfuegos, A. J. & Monelli, C. (1983) The testimony of political repression as a therapeutic instrument. *American Journal of Orthopsychiatry*, 53, 43–51.

Cohn, J., Holzer, K. I. M., Koch, L., *et al* (1980) Children and torture. *Danish Medical Bulletin*, 27, 238–239.

——, Danielsen, L., Holzer, K. I. M., *et al* (1985) A study of Chilean refugee children in Denmark. *Lancet*, *ii*, 437–438.

Goldfeld, A. E., Mollica, R. F., Pesavento, B. H., *et al* (1988) The physical and psychological sequelae of torture. *Journal of the American Medical Association*, 259, 2725–2729.

Gordon, E. & Mant, A. M. (1984) Examination of a teacher from El Salvador. *Lancet*, *i*, 213–214.

Horowitz, M. J. (1976) *Stress Response Syndromes*. New York: Jason Aronson.

Kinzie, J. D., Fredrickson, R. H., Ben, R., *et al* (1984) Post-traumatic stress disorder among survivors of Cambodian concentration camps *American Journal of Psychiatry*, 141, 645–650.

Krystal, H. & Niederland, W. G. (eds) (1971) *Psychic Traumatization. After Effects in Individuals and Communities*. Boston: Little, Brown & Co.

Levi, P. (1987) *If This is a Man; The Truce*. London: Sphere Books.

Lishman, W. A. (1987) *Organic Psychiatry*. Oxford: Blackwell Scientific.

Lunde, I., Rasmussen, O. V., Wagner, G., *et al* (1981) Sexual and pituitary–testicular function in torture victims *Archives of Sexual Behavior*, 10, 25–32.

Martin-Baro, I. (1988) From dirty war to psychological war. the case of El Salvador. In *Flight, Exile, and Return: Mental Health and The Refugee* (ed. A. Aron). San Francisco: Committee for Health Rights in Central America (CHRICA).

Mollica, R. F, Wyshak, G. & Lavelle, J. (1987) The psychosocial impact of war trauma and torture on southeast Asian refugees. *American Journal of Psychiatry*, 144, 1567–1572

Moses, R. (1978) Adult psychic trauma. The question of early predisposition and some detailed mechanisms. *International Journal of Psycho-Analysis*, 59, 353–363.

Niederland, W. G. (1968) Clinical observations on the survivor syndrome. *International Journal of Psycho-Analysis*, **49**, 313–315.

Pincus, J. H. (1978) Disorders of conscious awareness; hyperventilation syndrome. *British Journal of Hospital Medicine*, 312–313.

Rachman, S. (1980) Emotional processing. *Behaviour Research and Therapy*, **18**, 51–60.

Rasmussen, O. V. & Lunde, I. (1980) Evaluation of investigation of two hundred torture victims. *Danish Medical Bulletin*, **27**, 241–243.

—— & Marcussen, H. (1982) The somatic sequelae to torture. *Manedsskrift for Praktisk Laegerning*. (English version available from The International Centre for Rehabilitation of Torture Victims (RCT), Copenhagen.)

Ritterman, M. (1987) Torture, the counter therapy of the state. *Networker*, 43–47.

Schlapobersky, J. & Bamber, H. (1988) Torture as the perversion of a healing relationship; rehabilitation and therapy with the victims of torture and organised violence. Paper presented to the Annual Meeting, American Association for the Advancement of Science (copies available from the Medical Foundation, London).

Stover, E. & Nightingale, E. O. (eds) (1985) *The Breaking of Bodies and Minds*. New York: W. H. Freeman.

Svendsen, G. (1985) When dealing with torture victims social work involves the entire family. *Socialradgiveren*. (English version available from The International Rehabilitation Centre for Torture Victims (RCT), Copenhagen.)

Turner, S. W. (1989) Working with survivors. *Psychiatric Bulletin*, **13**, 173–176.

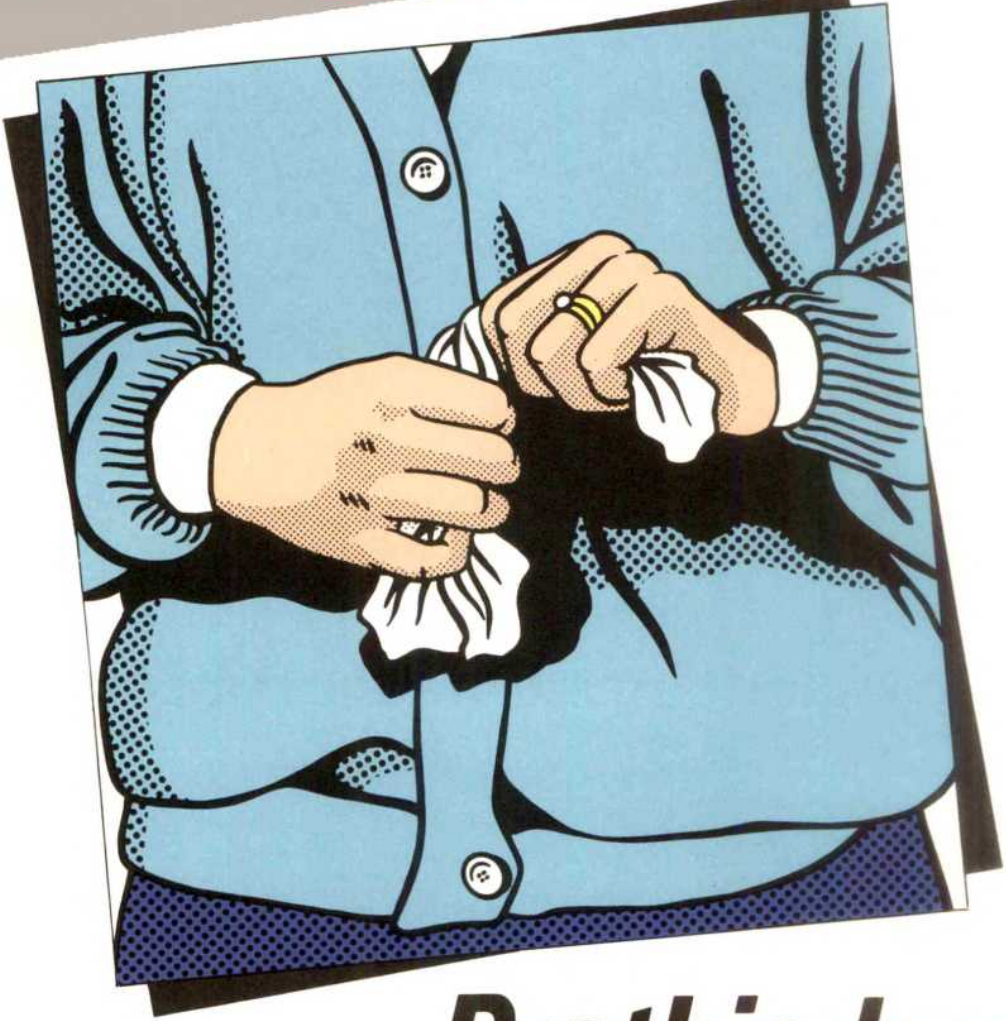
United Nations (1984) *Convention Against Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment*. New York: Office of Public Information, United Nations.

Wirtz, P. W. & Harrell, A. V. (1987) Effects of postassault exposure to attack-similar stimuli on long-term recovery of victims. *Journal of Consulting and Clinical Psychology*, **55**, 10–16.

*Stuart Turner, MA, MD, MRCP, MRCPsych, *Senior Clinical Lecturer in Psychiatry, University College and Middlesex School of Medicine, Wolfson Building, Middlesex Hospital, London W1N 8AA, and Consultant Psychiatrist, Medical Foundation for the Care of Victims of Torture*; Caroline Gorst-Unsworth, MB, ChB, MRCPsych, *Psychiatrist, Medical Foundation for the Care of Victims of Torture, 96–98 Grafton Road, London NW5 3EJ, and Research Associate, University College and Middlesex School of Medicine*

*Correspondence

"I just can't cope"



Prothiaden

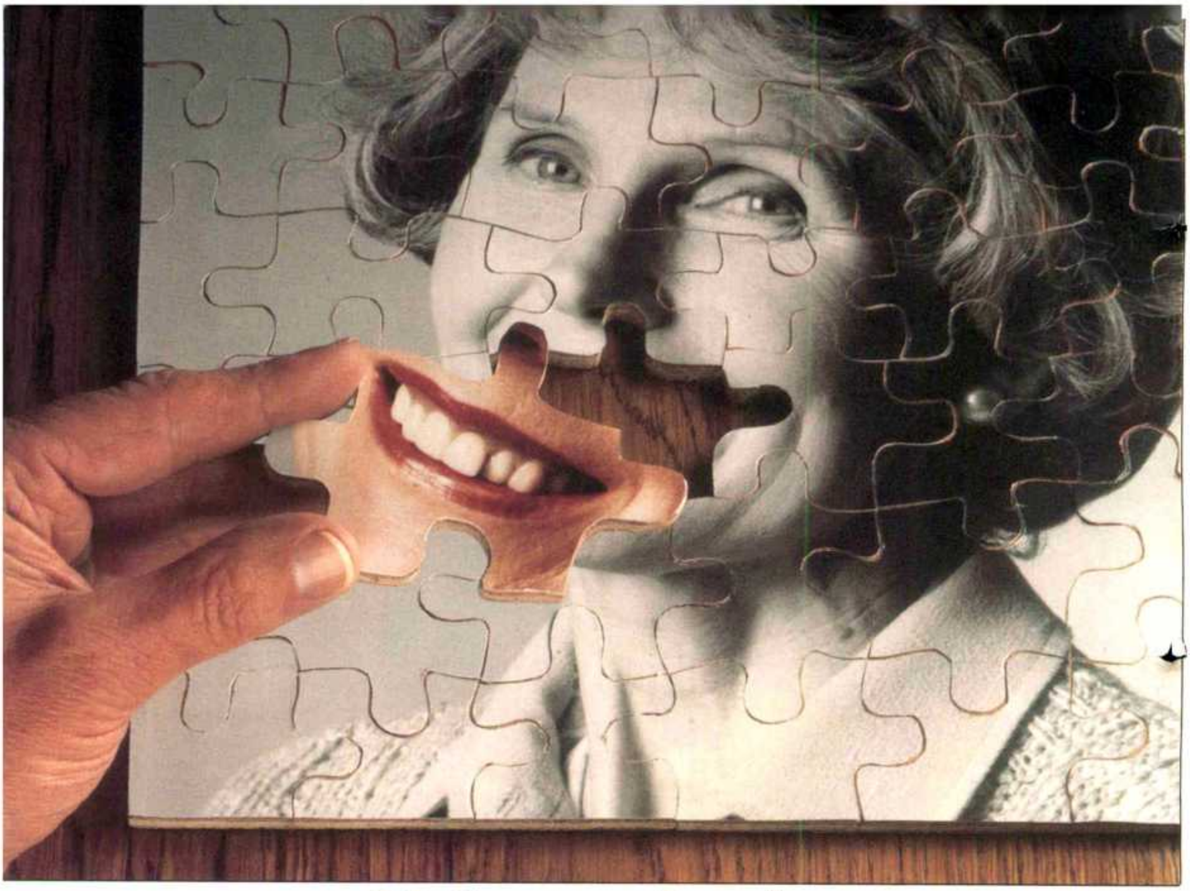
Dothiepin Hydrochloride B.P.

BOOTS PHARMACEUTICALS

TOMORROW'S STANDARD TODAY

PRESCRIBING INFORMATION Presentation: Prothiaden is Dothiepin Hydrochloride B.P. an antidepressant of the tricyclic group. It is available as 25mg capsules and 75mg tablets. **Uses:** Prothiaden is indicated in the treatment of depression and associated anxiety. **Dosage:** Prothiaden should be given in a dosage of 75 to 150mg daily, based on response and severity. This may be taken as a single evening dose. In certain circumstances, i.e. in hospital use, Prothiaden has been given in dosages up to 225mg daily. Elderly – half the normal dose may be sufficient. **Precautions and Contra-indications:** Prothiaden has anticholinergic properties, therefore, it may precipitate urinary retention in susceptible individuals and its use should be avoided in patients with existing or potential urinary retention. Prothiaden should not be used in patients with closed-angle glaucoma. Caution is advised when treating epileptic patients and those with cardiovascular disorders. Prothiaden should not be used with MAO inhibitors nor within 14 days of ceasing such treatment. It may modify the action of some CNS depressants, catecholamines and hypotensive agents. **Side Effects:** These are usually mild and normally controlled by reducing the dosage. The following have been reported: dryness of mouth, constipation, disturbed accommodation, orthostatic hypotension, palpitations, somnolence, tremor, headache. **Basic N.H.S. Price:** 28 × 75mg £4.00 as at 1st Oct. 1987. **Product Licence Numbers:** PL0014/5941, PL0014/0209. Boots Pharmaceuticals, Nottingham, England. Further information available on request.

ETH 1324/2/90



Help put back what depression takes out

averin is as effective as you might expect any
-line antidepressant to be.

ut Faverin is also generally non-sedating, so
er depressed patients are not affected by the
wsiness commonly associated with traditional
depressants.

nlike tricyclics, Faverin produces few
cholinergic effects which can add to the
blems of ageing, like constipation and blurred
on.

Nor does Faverin produce any clinically significant change in blood pressure or heart rate.

And – importantly – there have been no deaths attributable to Faverin alone in intentional or accidental overdosage up to 8600mg.

NEW SIMPLER DOSAGE

Faverin is now available in a new 100mg tablet size, which means a simpler dosage regimen – just one Faverin 100mg at night.

FAVERIN®

fluvoxamine ▼

ets the older depressed patient get on with lif

rin Prescribing Information ▼ Presentation: Round, yellow, enteric-coated
each containing 50mg fluvoxamine maleate, imprinted with 'Duphar 291', in packs of 60
basic NHS price £25.00. PL 0512/0070. Oval, yellow, enteric-coated tablets each containing
fluvoxamine maleate, imprinted with 'Duphar 313', in packs of 30 tablets, basic NHS price
. PL 0512/0072. **Uses:** The treatment of symptoms of depressive illness. **Dosage and
istration:** The tablets should be swallowed without chewing and with water. *Adults, including
erly:* Recommended starting dose of 100mg in the evening. Adjust according to response to
um of 300mg daily, in divided doses. *Children:* Not recommended. **Contra-indications,
ngs, etc:** Avoid during pregnancy and in nursing mothers, unless compelling reasons. Do not
h, or within two weeks of, ending treatment with monoamine-oxidase inhibitors. Faverin has
dose initially with careful monitoring. Discontinue if associated with increased hepatic enzymes.
effects of alcohol may be potentiated by Faverin. Improvement may be delayed for two or more we
and close monitoring is advised during this period. **Drug interactions:** Clinically signifi
interactions possible with drugs metabolised by liver and having narrow therapeutic index
warfarin, phenytoin and theophylline). Increased plasma levels of propranolol and warfarin see
practice. No interactions seen with atenolol or digoxin. **Side-effects:** Most commo
gastrointestinal; nausea, vomiting and diarrhoea. In some patients who report nausea, vomiting
occur. Caution should therefore be exercised when administering Faverin to patients with a mec
condition likely to be exacerbated by vomiting. Others include dizziness, somnolence, agitat
headache and tremor. Anxiety reported less often. Convulsions have been reported. Avoid in pati

British Journal of Psychiatry (1990), **157**, 481–490

Lecture

Structure and Function in Neurology and Psychiatry

E. H. REYNOLDS

In the 19th century the triumphs of neuropathology and the clinico-anatomical method led to the evolution of neurology as a separate 'organically' based discipline associated with the concept of functional localisation. At the same time the growth of psychodynamic psychiatry contributed to the progressive separation of the two disciplines, with neuropsychiatry sitting uneasily in the middle. Psychiatrists are now showing increasing interest in the structure and function of the nervous system, but are having difficulty in integrating their findings into 'functional' diseases. This may be because disorder of function in the nervous system is much more complex than previously envisaged. The function of the nervous system is profoundly affected by psychological and social factors. The view that neurology is wholly 'organic' and synonymous with structural disease of the nervous system is fallacious. Neurological patients have complex dynamic disorders of function in the nervous system whether or not structural disease is present.

> "The antithesis between 'organic' and 'functional' disease states still lingers at the bedside and in medical literature, though it is transparently false and has been abandoned long since by all contemplative minds."
> (Kinnier Wilson, 1940)

Neurological practice is rooted in structure, in neuroanatomy and neuropathology. One of the primary objectives of the neurologist is to find the lesion. For this he has developed his skills in neurological examination and utilises increasingly sophisticated and costly investigative techniques. Neurological disorder is widely referred to as organic. Yet structural lesions produce their symptoms and signs by disturbing the function of the nervous system: by interruption, decrease, increase or release of function; by excitation, inhibition, instability or restoration of function. However, this disturbance of function in neurology is often taught and practised as though it were a static or almost wholly 'reflex' neurophysiology – fixed lesions producing fixed defects in function. The neurological textbooks are littered with diagrams, figures and tables showing how a lesion at this or that site produces a clear-cut constellation of symptoms and signs. The reality for the observant neurologist is often rather different. Not all the expected symptoms and signs are present. Those that are present, whether motor, sensory, reflex, cognitive or behavioural, come and go or fluctuate in intensity over minutes, hours or days under a variety of intrinsic or extrinsic influences. Some are very intermittent, such as the seizures associated with a cerebral tumour or scar, or the migrainous headaches associated with an arteriovenous malformation. Furthermore, the symptoms and signs of a fixed structural lesion may be indistinguishable from those of functional disorders in the nervous system, for example the hemiparesis of a stroke and the Todd's paralysis following seizures. Psychological and social factors frequently clearly influence and interact with neurological symptoms and signs, but are often overlooked or ignored as though this was somehow part of 'psychiatry'.

Psychiatrists in general adopt an uncertain, even confused, approach to structure and function. Individually they may belong to very different schools of thought depending whether they are orientated to the neuropsychiatric or psychodynamic wing of the discipline (Hill, 1964). Some are concerned that they may be overlooking a structural lesion, something that really belongs to neurology! All recognise that various types of cerebral pathology produce mental symptoms which form part of the practice of psychiatry, so-called 'organic psychiatry' or 'neuropsychiatry' (Lishman, 1978; Trimble, 1981), which clearly has close links with neurology. At the very least it is borderline territory, the interface or bridge between the two disciplines. The main corpus of psychiatry is concerned with 'functional' disorder of the mind, self or psychic apparatus, brought about mainly by intra- and interpersonal forces of a psychological and social kind. Here the use of the word 'functional' is not

---

Presented to the symposium on the "Interface Between Neurology and Psychiatry" at the Royal College of Psychiatrists' meeting, October 1988; and a contribution to *The Bridge between Neurology and Psychiatry* (1989) (eds E. H. Reynolds & M. R. Trimble). Edinburgh: Churchill Livingstone.

physiological but psychological. The psychoses illustrate the dilemma, with the organic psychoses rooted in nervous system pathology and physiology, and the 'functional' psychoses conceived of in psychic formulations.

In recent years the conceptual dilemma for the psychiatrist has been made more acute by developments in the neurosciences, especially in psychopharmacology, neurochemistry and brain-imaging techniques. More and more attention is being paid by psychiatrists to these developments, which have often revealed unsuspected structural or functional (in the neurological sense) disorders in their patients. For neurologists I would also argue that a wholly organic approach, which ignores or overlooks the impact of psychological and social factors in neurological disorders, is incomplete and, from the patient's point of view in particular, inadequate. The present widely held concept of neurology as organic and psychiatry as 'functional', with neuropsychiatry sitting uneasily between, is unsatisfactory.

These contrasting views of structure and function by neurologists and psychiatrists have not come about by chance, but have been shaped by forces which have led to the separation of neurology and psychiatry as distinct disciplines with ill-defined borders. If we are to understand the present situation and to see our way foward to a more satisfactory and less confusing conceptual framework it is important to understand what these forces and developments were.

Historical background

In the beginning there were no neurologists or psychiatrists, but there were 'nervous diseases'. According to Lopez-Piñero (1983), the concept of 'nervous diseases' emerged in Britain with Willis (1622–75) and Sydenham (1624–89). For example, both viewed hysteria and hypochondria as nervous diseases and not, as in the Galenic tradition, vapours emanating from the uterus or the spleen, liver and stomach. According to Willis (1682), hysteria "the so-called uterine disease is primarily a convulsive disease caused by an alteration of the nerves and the brain". The concept of nervous diseases was consolidated in the 18th century, when they also became fashionable, due to the influence of, among others: Cheyne (1733), whose treatise on the "English malady" suggested that "these nervous diseases being computed to make almost one-third of the complaints of the people of condition in England"; and of Whytt (1765), who expressed the opinion that "all diseases may, in some sense, be called affections of the nervous system, because in almost every disease, the nerves are more or less hurt; and in consequence of this, various sensations, motions and changes are produced in the body".

It is to Cullen (1710–90) that we owe the term 'neurosis'. William Cullen succeeded Robert Whytt to the Chair of the Practice of Physic at Edinburgh. For Cullen the word 'neuroses' was no more than a neologism for Whytt's 'nervous diseases'. Cullen recognised four classes of disease: three were general (I pyrexiae, II neuroses and III caquexiae), the fourth local (IV locales). The class 'neuroses' was defined as "affections of sense and motion without fever or evidence of local disease", that is, they were general and functional affections of the nervous system and the presence of any local lesion would exclude them from the class. In his subclassification (below) of the neuroses, we see some disorders which belong to modern neurology (convulsions, paralysis, etc.) and some to modern psychiatry (hysteria, melancholia, etc.):

Comata (e.g. apoplexy, paralysis)
Adynamiae (e.g. syncope, hypochondriasis)
Spasmi (e.g. convulsions, chorea, hysteria, tetany)
Vesaniae (e.g. melancholia, mania, impaired judgement)

The concept of neuroses was taken up in France by Pinel (1725–1826), who translated Cullen's textbook. He, too, described neuroses as alterations of sensitivity and motility which are not accompanied by fever, inflammation or structural lesion (Pinel, 1798). His classification of the neuroses (below), like Cullen's nosology, contains a mixture of modern neurology and psychiatry, as well as some general medical disorders undoubtedly influenced by the nervous system:

Neuroses of the senses (e.g. amaurosis, diplopia, deafness, tinnitus)
Neuroses of cerebral function (e.g. coma, hypochondria, amentia, catalepsy, melancholia, mania, hydrophobia)
Neuroses of Locomotion (e.g. convulsions, chorea, palsy, tetanus, aphonia)
Neuroses of nutrition (e.g. dyspepsia, asthma, palpitations, colic, anorexia)
Neuroses of sexual function (e.g. priapism, hysteria)

The clinico-anatomical method set limits to what could be included under the class neuroses. As the discovery of any structural lesion by definition excluded a disorder from this category, the stage was now set for the systematic contraction of the neuroses. More and more disorders were eliminated by the progressive triumphs of French and other European neuropathologists and cerebral localisers throughout the 19th century, culminating in the great achievements of Charcot (1825–93) at the Salpêtrière. At the same time rearrangements of the classification of the neuroses on anatomical principles were introduced, such as that of Jaccoud (1872):

Brain neuroses (e.g. mental diseases)
Cerebrospinal neuroses (e.g. epilepsy, hysteria, catalepsy)
Brain-stem neuroses (e.g. chorea, tetanus, paralysis agitans)
Peripheral neuroses (e.g. anaesthesias, neuralgias, hyperkinesias)

The point remained, however, that these were all functional disorders of the nervous system for which there was no known structural pathology. If structural pathology was discovered in a neurosis it was either reclassified or sometimes a certain ambiguity crept in with the use of such phrases as 'symptomatic neurosis', for example idiopathic and 'symptomatic' epilepsy.

The same forces in the 19th century which were leading to the contraction of the neuroses were also leading to the growth of neurology as an independent scientifically based discipline. Thus the first textbook of neurology from Germany by Romberg (1840–46) was based on physiological or functional principles. Nervous diseases were classified into two main divisions: neuroses of sensitivity and neuroses of motility. However, by the end of the century neurological textbooks were firmly rooted in the new anatomy and pathology and to a much smaller extent on physiological principles, that is, on structure more than function. Furthermore the new neurophysiology was based on the principle of functional localisation following the pioneering studies of, for example, Broca (1865) and Wernicke (1874) with respect to speech and Ferrier (1878) with respect to motor and sensory functions. This new functional approach was however of a rather static kind, with lesions interrupting activities or pathways with fixed functions. Gowers' *Manual of Diseases of the Nervous System* published in 1886 illustrates the many new neurological diseases that had been discovered and classified on the basis of the new principles of neuropathology and localisation of function. Neurological textbooks, such as Gowers', still included functional disorders (i.e. neuroses), but exemplified the great reduction that had taken place in the number of neuroses of sensation and motility since Cullen, Pinel and Romberg.

Before turning to parallel developments in 19th-century psychiatry, it is appropriate to refer to the views of Hughlings Jackson (1835–1911). He accepted that some disorders of the nervous system were *functional* (i.e. were neuroses), including some insanities, which he linked to current evolutionary theories, proposing the concept of dissolution of nervous system function. However, Jackson recognised two kinds of abnormal function. As the normal function of nerve tissue was to "store up and expend force", so abnormal function could be either (a) loss of function (i.e. reduced or absent storage and expenditure), or (b) overfunction (i.e. increased storage and expenditure), the latter also contributing to *instability* of function. The former could lead to palsies and anaesthesias, the latter to epilepsies, chorea and tetanus. He believed that both had a pathological basis. However, although the "destroying lesions" responsible for the former were usually readily enough apparent to the pathologist, this was often not the case for his "discharging lesions", for which he had to invoke "morbid nutrition", for example in idiopathic epilepsy. Jackson emphasised that he was not using the word functional in the conventional 19th-century way of "absence of or undetected brain pathology" (Jackson, 1873).

Just as the number of neurological neuroses (functional disorders of the nervous system) was diminishing towards the end of the 19th century, a wholly new concept of the neuroses arose in the evolving discipline of psychiatry. Psychiatry has a longer history than neurology, as Hill (1964), among others, has described. Especially prominent throughout the 19th century was the German and Austrian school of neuropsychiatry, illustrated by Griesinger (1817–68), a contemporary of Romberg in Berlin, and Meynert (1833–92) in Vienna. For them, mental diseases were brain diseases. Meynert (1884) subtitled his psychiatric text book *Diseases of the Forebrain*. Neurosyphilis and general paralysis of the insane were the model. The French clinico-anatomical method was applied as vigorously to mental diseases as it was being applied to neurological diseases, although with less satisfactory results. Kraepelin (1856–25) belonged to this tradition, and so did Freud (1856–1939). Freud was well grounded in the new neuroanatomy, neuropathology and neurology, but he found these an incomplete explanation of the clinical phenomena he was observing, as his studies on aphasia made clear (Freud, 1891). As a young man he spent several months of 1885–86 at the Saltpêtrière in Paris, where he was greatly influenced by Charcot, who was now studying hysteria and hypnosis, which led him to suspect the presence of subconscious and psychological mechanisms. On his return to Vienna, Freud became more interested, not so much in how his patients spoke, but in what they said! Instead of brain disease, disorder of the psychic apparatus was to be understood and explained in relation to the new disciplines of psychoanalysis, psychology and sociology. Thus was born 20th-century dynamic psychiatry, which for the most part has tended to dominate 20th-century neuropsychiatry. This is illustrated by what has happened to the neuroses. With Freud and the dynamic psychiatrists appeared a whole new range of disorder and meaning – psychoneuroses (i.e. anxiety neurosis, obsessional neurosis, depressive neurosis, etc.). No longer disorders of sensation and motility to be understood on the basis of disturbance in nervous system physiology, but disorders of the psychic apparatus to be interpreted in terms of unconscious motivation or maladaption to psychological or social pressures or conflicts. Thus the words 'neurosis' and 'functional' were taken over by the dynamic psychiatrists with a new meaning. The growing numbers of neurologists and diminishing numbers of neuropsychiatrists were left with the word 'organic'.

In retrospect it is surprising that neurologists allowed psychiatrists to hijack the words 'neurosis' and 'functional' and invest them with new meaning without a protest. Kinnier Wilson (1878–1937) is distinguished, among other reasons, for the discovery of the disease which bears his name and for his textbook of neurology which was published in two volumes in 1940 after his death. He was the last British neurologist to include a section on 'the neuroses' in a textbook of neurology, and his classification can be summarised as:

Motor neuroses (e.g. tics, rhythmias, myospasms, myotonia, torticollis, occupational)
Sensory neuroses (e.g. neuralgias (e.g. trigeminal), causalgia, acroparaesthesias)
Reflex neuroses (e.g. eneuresis, retention)
Psychoneuroses (e.g. obsessional, anxiety)

In keeping with 19th-century neurological tradition, he recognised motor, sensory, reflex and psychoneuroses. Although he does not discuss psychoneuroses, as these belong to psychiatry, he does protest that to equate 'psychoneurosis' and 'neurosis' is to take a part for the whole. He goes on to maintain that "no radical distinction can exist between a 'neurosis' and any other type of nervous syndrome. In theory and by etymology it should denote disturbance of the intrinsic function of nerve tissue".

Kinnier Wilson understood that current neurological opinion took the word 'neurosis' to signify a disorder of nervous function for which, as yet, no underlying structural basis had been found. However, he pointed out that "molecular change must accompany all reactions whether those of a neurosis or of structural disease and, if on the one hand it induces lesions that can be seen and in the other not, no fundamental separation between them is thereby effected". He argues persuasively for the retention of the words neurosis and functional in the neurological camp. For him neurological symptoms, whether in the presence of structural disease or not, exhibit a dynamic quality which can only be explained by a dynamic neurophysiology, a functional disturbance in the nervous system which owes something to Jackson's views, as well as newer concepts of excitation and inhibition. Psychiatrists can have their psychoneuroses and explain them as they wish, but if they take over the words 'neurosis' and 'functional' completely, then presumably new words would have to be invented to describe the disturbances in nervous system function observed and treated by neurologists. He would have been disappointed to learn that the antithesis between 'organic' and 'functional', which he regarded as false, is even more deeply rooted today, as Trimble (1982) has also deplored. Other recent authors who have discussed changes in meaning which have occurred to the words 'organic' and 'functional' since the 19th century include Jeannerod (1985), Harrington (1987), and Clarke & Jacyna (1987).

The present scene

I have described how two processes in particular led to the progressive evolution of neurology and psychiatry as independent disciplines: on the one hand, the triumphs of neuropathology and the clinico-anatomical method culminated in the progressive replacement of the earlier functional neurology (i.e. disorders of sensation and motion) by a structurally based discipline associated with a rather fixed functional localisation; on the other hand, the evolution of dynamic psychiatry took with it the concepts of functional disorder and neuroses (nervous diseases) and gave them a new meaning within the conceptual framework of a psychic apparatus either separated from or only obscurely linked to the nervous system. Two consequences of these developments have been that: (a) psychiatry has tended to split into two camps with its neuropsychiatric wing having common roots and links with neurology; and (b) neurologists have tended either to overlook those complex disorders of function in the nervous system, which did not seem to fit into their more simplified views of functional localisation, or even to pass them off as in some way 'psychiatric'.

Two modern trends highlight the difficulties posed by a predominantly organic, structurally based and functionally localised neurology and a predominantly 'functionally' based psychiatry: (a) in neurology there is growing evidence that the disorders of function in the nervous system associated with structural disease, or even unassociated with structural disease, are much more complex and dynamic than hitherto understood; and (b) in psychiatry the discovery of structural lesions by modern imaging techniques are proving very difficult to integrate into 'functional' disorders.

Disorder of function in neurology

Excitability of the cerebral cortex

Function within the nervous system is achieved by "transient electrical potentials travelling the fibres of the nervous system" (Sherrington, 1941). Some insight into the functioning of the human cerebral cortex in health and disease was gained by Penfield's studies of cortical stimulation in patients with epilepsy undergoing evaluation for surgery (Penfield & Jasper, 1954; Penfield, 1958). He showed that functional activity in the cortex was not simple, repeatable and fixed, but complex, variable and dynamic. As in animals, functional localisation does not exist in 'centres' or 'points' but in areas and patterns that extend into various regions of the brain. Although the order of cortical representation of function across, for example, the motor or sensory cortex may be consistent from patient to patient, the site and size of representation may not only vary between patients but even within patients at successive explorations. As also noted by Grunbaum & Sherrington (1901, 1903) in anthropoids there is considerable variation in the degree to which an individual's cortex can be stimulated. Some variation is due to either facilitation or extinction, depending on the time between successive stimulations. However, the character and intensity of a response are also influenced by stimulation of adjacent points, implying that response will vary with the internal or external environment. Instability of response is a characteristic of the human cortex (Penfield & Welch, 1949). A motor or sensory response can be displaced into a previously unresponsive territory by advancing stimulations. Sometimes motor responses can be elicited from the sensory cortex or sensory responses from the motor cortex.

Seizure discharges often sensitise the cortex ('epileptic sensitisation') so that evidence of the functional activity of a particular area may be elicited by electrical stimulation, where usually no response would be found. For example, the temporal cortex, which ordinarily gives no psychical response to stimulation may, in the case of an epileptic patient, produce hallucinations or illusions, but only when that part of the cortex has been the seat of epileptic discharges. This activation does not necessarily apply only to patterns of response which appear in the patient's seizures, but can involve neighbouring elements of the cortex and other patterns of response. On other occasions after a seizure the cortex may be in a refractory state and this may be associated with displacement of responses to other areas of cortex. Depression of sensitivity to stimulation of one area may be associated with hypersensitivity of another area.

Disorder of function in epilepsy

The study of epileptic patients provides other evidence of dynamic disorder of function in neurological disease (Trimble & Reynolds, 1986). Even in the presence of an obvious cerebral lesion it has to be asked why seizures occur in some patients but not in others, and why seizures occur in an individual patient at particular times and not at others. It is often apparent that provocative factors, especially of a sensory or emotional kind, play a triggering role. Sometimes it is the summation of several factors that triggers an individual attack: anything that may increase the instability around Jackson's 'discharging lesion' (e.g. change in level of alertness or sleep, physical or emotional stress or fluctuation in hormonal pattern associated with menstruation) may precipitate a seizure.

Other considerations relating to the effects of individual seizures on brain function may also be important. It is apparent that most epileptic patients are well controlled on treatment or go into spontaneous remission (Reynolds *et al*, 1983). About a quarter, however, go on to develop chronic epilepsy apparently resistant to medication. How does this come about? Certain factors increase the risk of chronic epilepsy, such as brain damage, neurological, psychological and social handicaps. In addition, the longer seizures continue after the start of treatment the less likely they are to remit (Elwes *et al*, 1984). This is in keeping with Gowers' (1881) hypothesis that each seizure increases the predisposition to the next one. The natural history of untreated epilepsy is largely unknown, but in a retrospective study of new referrals with between two and five untreated tonic-clonic seizures it was remarkable that, in most patients, the time interval between successive seizures was shortening, suggesting an escalation of epilepsy (Elwes *et al*, 1988). All this suggests that early effective treatment may be important to prevent the evolution of chronic epilepsy (Reynolds, 1987, 1988). Epilepsy should perhaps be viewed not as a random succession of seizures but as a process – a process in which important events occur in the brain between seizures and in which the early course of the disease influences the later prognosis (Reynolds *et al*, 1983; Reynolds, 1989). It should be clearly understood that there are processes in the brain which lead to *remission* of epilepsy as well as escalation of the disorder.

Kindling

An experimental model which is to some extent relevant to Gowers' view of seizures begetting seizures and Penfield's observations on 'epileptic sensitisation' of the cortex is that of kindling (Goddard, 1967; Wada, 1976, 1981; Reynolds, 1989). In this model repeated subthreshold electrical or chemical stimuli progressively increase convulsive response, eventually culminating in a seizure. The subsequent application of a single subthreshold stimulus will again evoke a seizure. Eventually chronic seizures can be produced *without evidence of tissue damage*. There are variations of the model depending on the species, the nature of the stimulus, and the area of the brain to which it is applied, but a central feature is that every one of the stimuli are necessary to kindle the seizure, provided they are applied at sufficient intervals. In other words each stimulus leaves its mark on the brain, but the nature of this mark has so far eluded detection. If a subthreshold stimulus can change brain function, why not a seizure itself? Indeed, it is certain that some functional change takes place, as evidenced by the clinical effect of convulsive therapy in depression.

The mirror focus

Another model which remarkably illustrates the remote functional effects of a structural lesion in the brain is the mirror focus (Morrell, 1969). The mirror focus is a new epileptogenic focus in the same area of brain but contralateral to the original focus. The secondary focus is induced by and can become completely independent of the original epileptogenic focus. Again, the time scale of the phenomenon varies in different species and is longest in man. Morrell (1985) has marshalled the evidence, derived from studies of patients evaluated and treated surgically for unilateral temporal lobe lesions, that the phenomenon occurs in humans. Successful temporal lobectomy may be undertaken in patients with a unilateral structural lesion despite bitemporal epileptogenic discharges if the contralateral discharges have not yet become independent.

The influence of environment and social factors on cerebral activity and structure

Aggression can be studied in animals by means of electrical stimulation of the brain. Offensive-defensive reactions have been related to many subcortical structures, extending almost continuously from forebrain to brain-stem and involving ventral septum, pre-optic area, amygdala, stria terminalis, anterior and posterior hypothalamus, posteroventral nucleus of the thalamus, tectal area, central grey, reticular substance and spinothalamic tract (Delgado, 1967). There is, however, much that is uncertain in the anatomical and functional relationships of these various areas. Furthermore, many of the studies have been in restrained (i.e. caged) lower animals and without regard to social situations. Delgado (1967) and Delgado & Mir (1969) have overcome many of these limitations by studying monkeys equipped with intracerebral electrodes in whom stimulation was achieved by means of radio stimulators. The animals formed part of an established colony with three or four other monkeys, so that individual and group behaviour could be studied in various free ranging and social situations. The full pattern of well organised, purposefully orientated aggression, adapted to changes in the environment and related to the animals' past experience, was evoked by stimulation of the nucleus ventralis posterolateralis of the thalamus and the pedunculus cerebellaris medius. Of particular interest is that this full effect was only evident when the social rank of the stimulated monkey was high and disappeared when the animal was in a subordinate position. Thus, when a monkey's social status was low, brain stimulation induced only increased motor activity and occasional threats, which provoked attacks against it. As social rank improved stimulated attacks against other animals increased. The effect of stimulation of the same cerebral point therefore differed in degree and had opposite social consequences, depending on hierarchical position. Like the studies of epilepsy previously described, these findings indicate that functional representation in the brain should not be expressed in absolute terms, but cerebral activity is in a dynamic state modified by environmental and social inputs.

Environmental factors not only modify the function of the brain but may also alter its *structure*. The infant or immature brain is especially vulnerable to such modification. Experimental studies have shown that early loss of visual stimulation in one eye, or disruption of the usual binocular stimulation, will lead to permanent change in the architecture of the occipital cortex (Wiesel & Hubel, 1965; Blakemore & Cooper, 1970). The clinical counterpart is the child with uncorrected strabismus. To avoid diplopia, visual impulses from the weak eye are suppressed and permanent blindness develops. This does not happen in the adult or mature nervous system. Similar effects of sensory deprivation have been described in other areas of the brain, and conversely an enriched and stimulating environment enhances the branching of cortical dendrites and the development of dendritic spines (Bennett *et al*, 1964; Volkmar & Greenough, 1972). These observations are probably relevant to the effects of early emotional and social deprivation on later mental health and behaviour, as illustrated in the early infant deprivation studies in monkeys (Harlow *et al*, 1971) and the behavioural imprinting investigations of ethologists (Lorenz, 1970).

The neurological examples described above illustrate that whether or not structural disease is present, disorder of function in the nervous system is much more complex and dynamic than is usually envisaged by such phrases as 'functional localisation'.

Structural disease in psychiatry

Computerised tomography in schizophrenia

There is no doubt that in the last decade there has been a remarkable rekindling of interest in structural brain disease in psychiatric disorders, stimulated by the development of non-invasive, increasingly sophisticated imaging techniques. Most attention has focused on schizophrenia, especially the chronic disease. Since the report by Johnstone *et al* (1976), many studies using computerised tomography (CT) have confirmed significant lateral ventricle enlargement and increased ventricular:brain ratio (VBR) in chronic schizophrenia compared with various control groups (Reveley, 1985; Owens *et al*, 1985), so much so that Farmer *et al* (1987) felt able to say that this finding "may now be regarded as probably the most replicable biological feature which investigations of the condition have yet revealed". Even so, not all studies have confirmed the finding. Smith & Iacono (1986) have compared the data in 14 positive studies and seven negative studies. The VBR was significantly lower in the control groups from the positive studies than in the controls from the negative studies. They concluded that the differences between the studies had more to do with the choice of controls than schizophrenic patients. Owen & Lewis (1986) have also analysed the choice of controls and suggest that some differences between control values can be accounted for by the fact that sometimes patients with conditions known to be associated with ventricular enlargement have been used, whereas in others there has been a selection bias against controls with large ventricles. Even if it is accepted that the association of ventricular enlargement with schizophrenia is a reliable finding it is certain that it is not specific for the disorder as similar observations have been reported in manic-depressive illness (Pearlson & Veroff, 1981; Standish-Barry *et al*, 1982).

The significance of ventricular enlargement in schizophrenia is an even more contentious issue. At first it was suggested that it might be related to cognitive impairment ("the dementia of dementia praecox"; Johnstone *et al*, 1978) or the 'negative' clinical features of a 'type 2' syndrome (Crow, 1980) but subsequent studies have not supported this hypothesis (Owens *et al*, 1985; Kalakowska *et al*, 1985; Farmer *et al*, 1987). Others have suggested a relationship to different clinical subtypes, for example paranoid schizophrenia (Nasrallah *et al*, 1982), but again this has not been confirmed (Farmer *et al*, 1987). The latter authors reported an association with Schneider's first-rank symptoms and a complex relationship with Feighner diagnostic categories. Several studies have examined the relationship between enlarged ventricles and the genetic background of schizophrenia. Reveley *et al* (1982, 1984) reported that, in monozygotic twins discordant for schizophrenia, the schizophrenic probands had significantly larger ventricles than the unaffected control twins. Furthermore, large ventricles were associated with the absence of a family history of 'psychiatric disorder'. The authors therefore suggest that schizophrenia can be categorised into a familial form with normal ventricles and a presumed genetic aetiology, and a non-genetic form associated with enlarged ventricles due to environmental insults to the brain. However, several non-twin studies have cast doubt on the proposed inverse relationship between enlarged ventricles and a positive family history of schizophrenia (Nasrallah *et al*, 1982; Owens *et al*, 1985; DeLisi *et al*, 1986; Farmer *et al*, 1987).

Other uncertainties remain. Do the reported CT abnormalities antedate or postdate the onset of schizophrenia? Are they static or do they evolve in parallel with the clinical picture? Do they predispose to schizophrenia? Are they an intrinsic aspect of the disorder or are they the result of schizophrenia and its treatment? Such questions can only be clarified in the light of longitudinal imaging studies over many years, which have not yet been adequately undertaken (Nasrallah *et al*, 1986; Scottish Schizophrenia Research Group, 1989). Do patients with CT abnormalities represent a subgroup with so-called organic as opposed to 'functional' schizophrenia? Kalakowska *et al* (1985) looked specifically at indices of 'organic dysfunction' in schizophrenia and found no correlation even between the indices themselves (i.e. enlarged ventricles, cognitive impairment and 'soft' neurological signs). Neurological signs were related to a history of developmental abnormalities, cognitive impairment to higher doses of current medication, and large ventricles to neither of these variables. This suggested that the three signs may be determined by different sets of factors. Furthermore there were no clinical differences between those with and without organic signs, but only evidence of a more unfavourable prognosis in the former (Williams *et al*, 1985).

Where in the brain is schizophrenia?

In addition to the more general and widespread abnormalities suggested by the CT findings, many other studies of a structural (CT, MRI, neuropathology) or functional (cerebral blood flow, psychometry, evoked potentials, PET and SPECT) kind have focused interest on specific brain regions as the site of the lesion or dysfunction in schizophrenia, for example the frontal lobes, the temporal lobes and limbic system, the left hemisphere, the basal ganglia, the corpus callosum, the hypothalamus, diencephalon and even the cerebellum (Andreasen, 1986). Indeed, we are in the midst of an explosion of interest in studies of this type. It seems that many authors regard the seat of the lesion or dysfunction in this disorder as one of the several sites described above on which they have focused their attention and investigations. By way of illustration, Weinberger *et al* (1986) and Berman *et al* (1986) have summarised their own and other evidence implicating the frontal lobes in schizophrenia and suggest a physiological dysfunction of the dorsolateral pre-frontal cortex to account for both the 'negative' and 'positive' features of the disease. A study using magnetic resonance imaging is said to be in keeping with the 'hypofrontality hypothesis' (Andreasen *et al*, 1986). Others have emphasised the importance of the temporal lobes in schizophrenia (Trimble, 1987).

The number and variety of attempts to localise schizophrenia in this or that region of the brain emphasises their implausibility, which stretches the credulity of a neurologist, who is used to seeing many patients without schizophrenia with lesions in the incriminated regions of the brain. These attempts to localise schizophrenia are in part based on what some psychiatrists view as a rediscovery of the writings and wisdom of 19th-century neurologists, with their emphasis on functional localisation. This renewal of interest in localised function in psychiatric disorders is sometimes referred to, especially in the USA, as behavioural neurology (Pincus & Tucker, 1985). However, disorder of function in the nervous system is now seen to be complex and dynamic. Is it conceivable that the complex disorders of neurological/psychological function which occur in schizophrenia can be understood in terms of only one system such as the dorsolateral pre-frontal cortex?

Conclusions

Two modern developments suggest the possibility of reconciling the logically confusing, if practically convenient, diverging trends in neurology and psychiatry. (a) The current widely held view that neurology is wholly 'organic' and somehow synonymous with structural disease of the nervous system is fallacious. Neurological patients have complex dynamic disorders of function in the nervous system, whether or not they have structural disease. These disorders of function cannot be understood wholly in terms of oversimplified notions of functional localisation. A single structural lesion may have remote functional effects far from the pathological site. Cerebral function is profoundly influenced by psychological and social factors. (b) In psychiatry the renewed interest in and discoveries of cerebral pathology with modern imaging techniques are proving difficult to integrate because they are being inappropriately linked to functional localisation. It need not be expected, for example, as seems to be a common view, that such hypothetical lesions or dysfunctions will be found in all cases of schizophrenia. Like epilepsy, schizophrenia may or may not be associated with cerebral pathology which may sometimes influence the course and prognosis. This need not lead to separate categories of organic and functional in the psychological sense. Both, however, remain functional disorders of the nervous system (neuroses) in the older, neurophysiological sense of the word, whether or not detectable pathology is present. Both are influenced by psychological and social factors. Why should one (epilepsy) be viewed as organic and the other (schizophrenia) as functional? Nor is the answer to reclassify schizophrenia as organic, as some demand. Psychiatrists might profitably spend less time taking sides in inappropriate conflicts between false dichotomies, and neurologists might usefully spend more time studying the influence of psychological and social factors on brain function and on their patients' functional disabilities.

In the 18th and 19th centuries, neurology was conceived in terms of disorders of 'sensation and motion', as a functional discipline. The difficulty at that time, for many historical reasons, was to know where to place disorders of the 'mind' in this scheme of things. In the event they were split into 'neuropsychiatry' and 'psychodynamic psychiatry' (Hill, 1964). It is not difficult today to envisage the mind with its varied activities (attention, memory, mood, etc.) as a function of the brain, like sensation and motion. This in no way undermines the importance of psychological, social or psychodynamic factors in either psychiatric or neurological disorders, but it does profoundly alter our conception of both types of disorder and the relationship of the two disciplines.

References

Andreasen, N. C. (ed.) (1986) *Can Schizophrenia be Localized in the Brain?* Washington, DC: American Psychiatric Press.

——, Nasrallah, H. A., Dunn, V , *et al* (1986) Structural abnormalities in the frontal system in schizophrenia: a magnetic resonance imaging study. *Archives of General Psychiatry*, **43**, 136–144.

Bennett, E., Diamond, M. C., Krech, D., *et al* (1964) Chemical and anatomic plasticity of the brain. *Science*, **146**, 610.

Berman, K. F., Zec, R. F & Weinberger, D. R. (1986) Physiologic dysfunction of dorsolateral prefrontal cortex in schizophrenia. II. Role of neuroleptic treatment, attention, and mental effort. *Archives of General Psychiatry*, **43**, 126–135.

Blakemore, C. & Cooper, G. F. (1970) Development of the brain depends on the visual environment. *Nature*, **228**, 477–478.

Broca, P. (1865) Sur la facalte du langage articule. *Paris Bulletin Societe Anthropologie*, **5**, 337–393.

Cheyne, G. (1733) *The English Malady: or, A Treatise of Nervous Diseases of all Kinds, as Spleen, Vapours, Lowness of Spirits, Hypochondriacal and Hysterical Distempers*. London: Strahan & Leake.

Clarke, E. & Jacyna, L. S (1987) *19th Century Origins of Neuroscientific Concepts*. Berkeley: University of California Press.

Crow, T. J. (1980) Molecular pathology of schizophrenia: more than one disease process. *British Medical Journal*, **280**, 66–68.

Cullen, W. (1769) *Apparatus ad Nosologiam Methodicam, seu Synopsis Nosologiae Methodicae in Usum Studiosorum*. Edinburgh: Creech.

—— (1777) *First Lines of the Practice of Phsyic*. Edinburgh: Creech

Delgado, J. M. R. (1967) Aggression and defense under cerebral radio control. In *Aggression and Defense, Neural Mechanisms and Social Patterns* (eds C. D Clemente & D. Lindsley) UCLA Forum in Medical Science, no. 7, vol. V., pp. 171–193. Berkeley: University of California Press.

—— & Mir, D. (1969) Fragmental organization of emotional behaviour in the monkey brain. *Annals of the New York Academy of Science*, **159**, 731–751.

DeLisi, L. E., Goldin, L. R. Hamovit, J. R., *et al* (1986) A family study of the association of increased ventricular size with schizophrenia. *Archives of General Psychiatry*, 43, 148–153.

Elwes, R. D. C., Johnson, A. L., Shorvon, S. D., *et al* (1984) The prognosis for seizure control in newly diagnosed epilepsy. *New England Journal of Medicine*, **311**, 944–947.

——, —— & Reynolds, E H. (1988) The course of untreated epilepsy. *British Medical Journal*, **297**, 948–950.

Farmer, A., Jackson, R., McGuffin, P., *et al* (1987) Cerebral ventricular enlargement in chronic schizophrenia: consistencies and contradictions. *British Journal of Psychiatry*, **150**, 324–330.

Ferrier, D. (1878) *The Localisation of Cerebral Disease*. London: Smith, Elder & Co.

Freud, S. (1891) *Zur Auffassung der Aphasien*. Wien. (Authorised translation with an introduction by E. Stengel (1953) London: Imago Publishing Company.)

Goddard, G. V. (1967) Development of epileptic seizures through brain stimulation at low intensity. *Nature*, **214**, 1020–1021.

Gowers, W. R. (1881) *Epilepsy and Other Chronic Convulsive Disorders: Their Causes, Symptoms and Treatment*. London: Churchill.

—— (1886) *Manual of Disorders of the Nervous System*. London: Churchill.

Grunbaum, A. S. F. & Sherrington, C. S. (1901) Observations on physiology of the cerebral cortex of some of the higher apes. *Proceedings of the Royal Society, London*, **69**, 206.

—— & —— (1903) Observations on physiology of the cerebral cortex of anthropoid apes. *Proceedings of the Royal Society, London*, **72**, 152.

Harlow, H. F., Harlow, M. K. & Suomi, S. J. (1971) From thought to therapy; lessons from a primate laboratory. How investigation of the learning capability of rhesus monkeys has led to the study of their behavioural abnormalities and rehabilitation. *American Science*, **59**, 538–549

Harrington, A. (1987) *Medicine, Mind and the Double Brain*. Princeton: Princeton University Press.

Hill, D. (1964) The bridge between neurology and psychiatry *Lancet*, *i*, 509–514.

Jaccoud, S. (1872) *Traite de Pathologie Interne* (2nd edn). Paris: Delahaye.

Jackson, H. J. (1873) On the anatomical, physiological and pathological investigation of epilepsies *Reports of the West Riding Lunatic Asylum*, **3**, 315–339.

Jeannerod, N. (1985) *The Brain Machine*. Cambridge, Massachusetts: Harvard University Press.

Johnstone, E. C., Crow, T. J., Frith, C. D., *et al* (1976) Cerebral ventricular size and cognitive impairment in chronic schizophrenia. *Lancet*, *ii*, 924–926.

——, ——, ——, *et al* (1978) The dementia of dementia praecox. *Acta Psychiatrica Scandinavica*, **57**, 305–324

Kalakowska, T., Williams, A. O. Ardern, M., *et al* (1985) Schizophrenia with good and poor outcome 1: Early clinical features, response to neuroleptics and signs of organic dysfunction. *British Journal of Psychiatry*, **146**, 229–246.

Kinnier Wilson, S. A. (1940) *Neurology*. London: Arnold.

Lishman, A. W. (1978) *Organic Psychiatry: The Psychological Consequences of Cerebral Disorder*. Oxford: Blackwell Scientific.

Lopez-Piñero, J. M. (1983) *Historical Origins of the Concept of Neurosis* (trans. D. Berrios). Cambridge: Cambridge University Press.

Lorenz, K. (1970) *Studies in Animal and Human Behaviour*, vol. 1 (trans. R. Martin). London: Methuen.

Meynert, T. (1884) *Psychiatrie: Klinic der Erkrankungen def Vorderhirns*. Vienna: W. Braumuller.

Morrell, F. (1969) Physiology and histochemistry of the mirror focus. In *Basic Mechanisms of the Epilepsies* (eds H. H. Jasper, A. A. Ward & A. Pope), pp. 357–370. Boston: Little Brown.

—— (1985) Secondary epileptogenesis in man. *Archives of Neurology*, **42**, 318–335.

Nasrallah, H. A., Jacoby, C. G., McCalley-Whitters, M., *et al* (1982) Cerebral ventricular enlargement in subtypes of chronic schizophrenia. *Archives of General Psychiatry*, **39**, 774–777

——, Olson, S. C., McCalley-Whitters, M., *et al* (1986) Cerebral ventricular enlargement in schizophrenia. *Archives of General Psychiatry*, **43**, 157–159.

Owen, M. J., Lewis, S. W. (1986) Lateral ventricular size in schizophrenia. *Lancet*, *ii*, 223–224.

Owens, D. C. C., Johnstone, E. C., Crow, T J , *et al* (1985) Lateral ventricular size in schizophrenia; relationship to the disease process and its clinical manifestations. *Psychological Medicine*, **15**, 27–41.

Pearlson, G. D. & Veroff, A. E. (1981) Computerised tomographic scan changes in manic depressive illness. *Lancet*, *ii*, 470

Penfield, W. (1958) The excitable cortex in conscious man. *The Sherrington Lectures*. Liverpool: Liverpool University Press.

—— & Welch, K. (1949) Instability of response to stimulation of the sensorimotor cortex of man. *Journal of Physiology*, **109**, 358–365.

—— & Jasper, H. (1954) *Epilepsy and the Functional Anatomy of the Human Brain*. London: Churchill

Pincus, J. H. & Tucker, G. J (1985) *Behavioural Neurology*. Oxford: Oxford University Press.

Pinel, P (1798) *Nosographie Philosophique, ou la Methode de L'Analyse appliquee a la Medicine*. Paris: Brosson.

Reveley, A. M., Reveley, M. A., Clifford, C. A., *et al* (1982) Cerebral ventricular size in twins discordant for schizophrenia. *Lancet*, *i*, 540–541.

——, —— & Murray, R. M. (1984) Cerebral ventricular enlargement in nongenetic schizophrenia; a controlled twin study. *British Journal of Psychiatry*, **144**, 89–93.

Reveley, M. A. (1985) Ventricular enlargement in schizophrenia: the validity of computerised tomographic findings. *British Journal of Psychiatry*, **147**, 233–240.

Reynolds, E. H. (1987) Early treatment and prognosis of epilepsy. *Epilepsia*, **28**, 97–106

—— (1988) Prevention of chronic epilepsy. *Epilepsia*, **29** (suppl. 1), S25–S28.

—— (1989) The process of epilepsy. In *The Clinical Relevance of Kindling* (eds T. Bolwig & M R Trimble), pp. 149–160. Chichester: Wiley.

——, Elwes, R. D. C. & Shorvon, S D (1983) Why does epilepsy become intractable? Prevention of chronic epilepsy. *Lancet*, *ii*, 952–954.

Romberg, M. H. (1840–46) *Lehrbuch der Nervenkrankheiten des Menschen*. Berlin: Duncker

Scottish Schizophrenia Research Group, MacDonald, H L & Best, J. J. K. (1989) The Scottish first episode schizophrenia study VI Computerised tomography brain scans in patients and controls. *British Journal of Psychiatry*, **154**, 492–498.

Sherrington, C S. (1941) Man on his nature *The Gifford Lectures, Edinburgh 1937–8* Cambridge. Cambridge University Press.

Smith, G. N. & Iacono, W. G. (1986) Lateral ventricular size in schizophrenia and choice of control group. *Lancet*, *ii*, 145.

Standish-Barry, H. M. A. S, Bouras, N., Bridges, P. K., *et al* (1982) Pneumoencephalographic and computerised axial tomography scan changes in affective disorder. *British Journal of Psychiatry*, **141**, 614–617.

Trimble, M. R. (1981) *Neuropsychiatry*. Chichester: Wiley.

—— (1982) Functional diseases. *British Medical Journal*, **285**, 1768–1770

—— (1987) The neurology of schizophrenia. In *Recurrent and Chronic Psychoses* (ed. T J Crow). Edinburgh· Churchill Livingstone.

—— & Reynolds, E. H. (eds) (1986) *What is Epilepsy?* Edinburgh: Churchill Livingstone.

Volkmar, F. R. & Greenough, W. J. (1972) Rearing complexity affects branching of dendrites in visual cortex of rats *Science*, **176**, 1445–1447.

Wada, J. A. (ed.) (1976) *Kindling*. New York: Raven Press.

—— (ed.) (1981) *Kindling 2*. New York: Raven Press

Weinberger, D. R., Berman, K. F. & Zec, R. F. (1986) Physiologic dysfunction of dorsolateral prefrontal cortex in schizophrenia. I. Regional cerebral blood flow evidence *Archives of General Psychiatry*, **43**, 114–124.

Wernicke, K. (1874) *Der Aphasische Symptomkomplex* Breslau: Kohn & Neigart.

Whytt, R. (1765) *Observations on the Nature, Causes and Cure of Those Disorders Which Are Commonly Called Nervous, Hypochondriac or Hysteric*. Edinburgh: Beckett & Du Hondt.

Wiesel, T. V. & Hubel, D. H. (1965) Extent of recovery from the effects of visual deprivation in kittens. *Journal of Neurophysiology*, **28**, 1029–1040.

Williams, A. O., Reveley, M. A., Kolakowska, T., *et al* (1985) Schizophrenia with good and poor outcome. II. Cerebral ventricular size and its clinical significance. *British Journal of Psychiatry*, **146**, 239–246.

Willis, T. (1682) *Opera Omnia*. Amstelodami: Wetstenius

E. H. Reynolds, MD, FRCP, FRCPsych, *Consultant Neurologist, Maudsley and Kings College Hospitals, Denmark Hill, London SE5 9RS*

British Journal of Psychiatry (1990), 157, 491–499

Review Article

Overexcitement and Disinhibition

Dynamic Neurotransmitter Interactions in Alcohol Withdrawal

PAUL GLUE and DAVID NUTT

In alcohol withdrawal, abnormalities occur in a number of neurotransmitter systems: there is reduced inhibitory function, and increased activity of excitatory systems. The former, indicated by reduced GABA and alpha-2-adrenoceptor activity, acts in conjunction with, and is exacerbated by, the latter, which itself may be due to the potentiation of NMDA activity by depletion of magnesium, and overactivity of catecholaminergic and CRF neurones. These dysfunctions produce immediate effects and may also contribute to the long-term changes in brain excitability by a kindling-like process. It is possible that early and active treatment may oppose this process. Present strategies for treatment of alcohol withdrawal enhance GABA and alpha-2 inhibitory, or reduce excitatory, mechanisms. Future possibilities include the use of CRF and/or NMDA antagonists.

The pathophysiology of alcohol withdrawal is incompletely understood. Disturbances have been reported in a number of measures, including electrolyte imbalances, altered neurotransmitter levels and receptor sensitivity, although these reports have tended to focus on only one or two areas of disturbance. In this article we attempt to integrate several of the different receptor and neurotransmitter abnormalities seen in alcohol withdrawal with the clinical symptoms and signs. These changes can be divided into those involving reduced inhibitory function and those related to increased excitation. They are represented diagrammatically in Fig. 1. From this information, more rational treatment strategies for alcohol withdrawal are proposed.

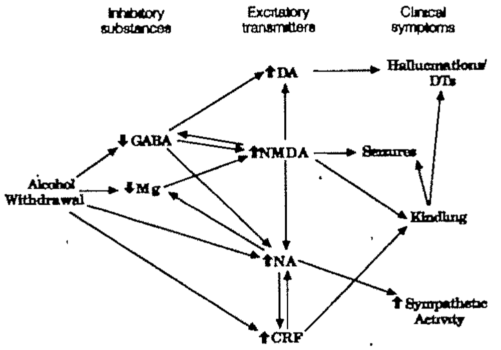


FIG. 1 Summary of receptor dysfunction in alcohol withdrawal: thin arrows (→) indicate the direction of relationships between alcohol withdrawal, inhibitory substances, excitatory transmitters and clinical symptoms; thick arrows indicate inhibition (↓) or excitation (↑) of receptor. (e g. alcohol withdrawal results in a low concentration of Mg. Overactivity of NMDA receptors, due to this low Mg, can induce seizures and accentuate NA or DA release.)

Reduced inhibitory input

Magnesium

Recent pharmacological investigators unrelated to alcohol research have thrown light on the association between low plasma magnesium (Mg) concentrations and propensity for seizures in alcohol withdrawal. The new understanding of the complexity of the excitatory amino acid/N-methyl-D-aspartate (EAA/NMDA) complex has revealed that activation of glutamate/NMDA receptors in brain areas associated with seizures (e.g. hippocampus) is accompanied by an increased cation flux into cells. This leads to increased cell firing, increased calcium entry, and possibly, if uncontrolled, intracellular damage or cell death (Garthwaite & Garthwaite, 1986). The cation channel gated by the NMDA receptor is partly controlled by the presence of Mg ions, which appear to occupy part of the ionophore (Nowak *et al*, 1984). Thus, Mg ions block the cation channel and oppose the effects of NMDA stimulation, and so are important endogenous inhibitory modulators. The Mg blockade of this channel is concentration-dependent and, in model systems, low Mg levels lead to paroxysmal firing of certain hippocampal cells (Herron *et al*, 1985*a*). We propose that the hypomagnesaemia of chronic alcoholism would lead to chronic excitatory activation (see Fig. 1), and so predispose to many of the symptoms of withdrawal, especially seizures and delirium tremens (DTs).

Levels of Mg in the plasma and cerebrospinal fluid (CSF) tend to be lower in chronic alcoholics than in normal controls (Flink, 1986). Alcohol ingestion causes increased Mg excretion in both alcoholics (Krystal, 1959) and normal subjects (Heaton *et al*, 1962). It is likely there is substantial depletion of whole-body Mg in alcoholics, because, after Mg administration, alcoholics have a net retention of Mg in contrast to normal subjects who have no retention (Flink *et al*, 1957). Acute cessation of drinking in chronic alcoholics causes a simultaneous fall in plasma Mg (Wolfe & Victor, 1969). This may be partly a response to the increase in plasma catecholamine concentrations (Whyte *et al*, 1987) seen in alcohol withdrawal (Potter *et al*, 1984).

Low levels of Mg have been associated with increased convulsive sensitivity to photic stimulation in alcoholics, which can be reversed by infusion of magnesium sulphate (Wolfe & Victor, 1969). The most severe withdrawal symptoms are seen in patients with the lowest Mg levels (Flink, 1986). Withdrawal seizures also occur more frequently in alcoholics with hypomagnesaemia (Wolfe & Victor, 1969), and can be reduced by Mg treatment (Daus *et al*, 1985).

Gamma-aminobutyric acid

Gamma-aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the central nervous system (CNS). Furthermore, the benzodiazepines also work through a receptor which facilitates GABA function. Although not seen in all studies, there is an extensive literature on the potentiation of GABA function by alcohol (Ticku *et al*, 1983). This can be seen in neuronal responses (Banna, 1969; Davidoff, 1973; Nesteros, 1980), receptor binding and chloride flux studies (Seilicovich *et al*, 1985; Suzdak *et al*, 1986*a*,*b*; Ticku & Rastogi, 1986; Ticku *et al*, 1986) as well as in behavioural models (Cott *et al*, 1976; Frye & Breese, 1982; Liljeqvist & Engel, 1982; Martz *et al*, 1983). Furthermore, alcohol may potentiate benzodiazepine receptor binding and function (Ticku & Davis, 1981; Ticku *et al*, 1983; Rottenburg, 1985). Recent studies have shown that a new class of benzodiazepine receptor ligand, the inverse agonists, will oppose many of the pharmacological effects of alcohol (Suzdak *et al*, 1986*a*; Lister & Nutt, 1987). Finally, a genetic component to GABA/alcohol interactions has been reported in certain mouse strains (Marley & Wehner, 1987). Clearly, this has marked implications for understanding the inheritance of alcoholism, and warrants evaluation in humans.

There is evidence that GABA/benzodiazepine receptor function is down-regulated after chronic alcohol administration, presumably in compensation for the enhancing effect of alcohol (Freund, 1980; Rottenberg, 1985; Rastogi *et al*, Allan & Harris, 1987). Such a down-regulation of GABA function could cause or contribute to the symptoms of alcohol withdrawal (Littleton, 1989). Consistent with this hypothesis, benzodiazepines (Cowen & Nutt, 1982), and more recently, selective GABA-agonist drugs such as progabide (Fadda *et al*, 1985) and gamma-hydroxybutyrate (Gallimberti *et al*, 1989), have been shown to reduce withdrawal symptoms. Furthermore, Lister & Karanian (1987) demonstrated that benzodiazepine inverse agonists exacerbate alcohol-withdrawal symptoms.

There are fewer clinical studies looking at GABA function in withdrawal. Cerebrospinal-fluid GABA levels during alcohol withdrawal and early abstinence are similar, although in the absence of a control group it is not possible to determine if they differ from normal (Hawley *et al*, 1981). In addition, chronic alcoholics have reduced plasma GABA levels (Coffman & Petty, 1985). Post-mortem study of alcoholics' brains has shown increased GABA receptor numbers (Tran *et al*, 1981). This could be an adaptive response to the potentiation of GABA inhibition by alcohol by altering receptor affinity states (see above).

GABA neurones have major inhibitory influences on monoamine and NMDA neurones. Activity of noradrenergic and dopaminergic neurones is reduced by GABA or by benzodiazepine agonists (Taylor & Laverty, 1969). Epileptiform activity in hippocampal cells caused by the GABA-A antagonist bicuculline is reduced by NMDA antagonists (Herron *et al*, 1985*b*), and after electrical kindling, reduced sensitivity to GABA and increased sensitivity to NMDA are seen (Stelzer *et al*, 1987). This would suggest that reduced GABA activity in alcohol withdrawal would lead to reduced inhibition of monoamine and NMDA neuronal activity; the increased NMDA activity may in turn further reduce GABA activity, leading to a vicious circle of progressively increasing excitation (see Fig. 1).

Increased excitatory input

Catecholamines: noradrenaline and dopamine

Alcohol has a complex effect on noradrenergic neurones (Nutt & Glue, 1986). However, noradrenaline (NA) overactivity is almost certainly present in alcohol withdrawal. Plasma and CSF NA and its major metabolite 3-methoxy-4-hydroxyphenylethylene glycol (MHPG) are increased by alcohol intoxication and withdrawal from single doses of alcohol (Borg *et al*, 1981). Much higher levels are seen during withdrawal from chronic intoxication (Potter *et al*, 1984; Hawley *et al*, 1985). The increase in NA activity may result from reduced alpha-2-adrenoceptor autoreceptor inhibition (Nutt *et al*, 1988*b*), which may persist into abstinence (Glue *et al*, 1989). Increased NA levels may aggravate hypomagnesaemia (see above), thus producing another positive feedback loop (see Fig. 1).

It is possible that dopamine (DA) dysfunction may be a contributing factor in the hallucinations of alcohol withdrawal, especially DTs (see Fig. 1). There is evidence of enhanced DA receptor sensitivity in the nucleus accumbens of rats following very long-term alcohol administration (Engel & Liljeqvist, 1976; Liljeqvist, 1978). Annunziato *et al* (1983) found endocrine evidence of DA receptor supersensitivity four to seven days after withdrawal. Whether these changes would lead to increased DA function in withdrawal, or merely reflect reduced DA activity, is a matter for further research.

Corticotrophin-releasing factor

This is a hypothalamic peptide that controls the release of adrenocorticotrophic hormone (ACTH) from the pituitary (Vale *et al*, 1981), but also is found in many other brain regions. Koob & Bloom (1985) suggested that the role of corticotrophin-releasing factor (CRF) is to co-ordinate stress responses at endocrine and behavioural levels. For instance, intracerebroventricular (i.c.v.) administration of CRF in rats causes changes suggestive of increased fear or anxiety (Koob & Bloom, 1985). However, CRF can also produce long-term behavioural changes, in particular seizures. Administration of CRF into the cerebral ventricles in rats induces seizures (Weiss *et al*, 1986) which resemble those caused by electrical kindling of the amygdala (Ehlers *et al*, 1983). Furthermore, pre-treatment with CRF sensitises to electrical kindling (Weiss *et al*, 1986).

Alcohol has several effects on CRF in animals. Acute exposure to alcohol appears to increase CRF production, whereas chronic alcohol reduces hypothalamic CRF content (Rivier *et al*, 1984), either by depletion or inhibition of synthesis (Dave *et al*, 1986). Despite this, behavioural responses to i.c.v. CRF, which are enhanced in rats given chronic alcohol, are further increased during alcohol withdrawal, and it has been suggested that this may reflect CRF-induced kindling (Ehlers & Chaplin, 1987).

Evidence for CRF dysfunction in alcoholics comes from the disruption of the hypothalamic–pituitary–adrenal axis in alcohol withdrawal. Excessive cortisol secretion has been reported (Valimaki *et al*, 1984). A detailed longitudinal study demonstrated that as well as marked hypocortisolaemia, the normal diurnal rhythm of cortisol is lost during this time (Risher-Flowers *et al*, 1988). These findings are consistent with CRF overactivity in withdrawal, a supposition which is supported by the blunted ACTH response to CRF in early abstinence (Adinoff, 1987; Holsboer *et al*, 1987; Heuser *et al*, 1988). Excessive CRF release would be expected to produce receptor subsensitivity, and the elevated cortisol levels should produce further inhibition (Adinoff, 1987).

Plasma concentrations of CRF in alcoholics have not been studied as the concentrations of this hormone in peripheral blood are below the sensitivity of present assay methods. CRF concentrations have been measured in the CSF of recently detoxified alcoholics, and were similar to those of normal controls (Nutt *et al*, 1988*a*). However, this has not been studied in acute withdrawal, which would be more relevant.

N-methyl-D-aspartate

N-methyl-D-aspartate (NMDA) receptors are increasingly recognised as having a major influence in synaptic transmission in the brain (Watkins & Evans, 1981). They have been implicated in long-term potentiation (LTP) of hippocampal neurones (Collingridge & Bliss, 1987), a process whereby repetitive stimulation produces long-lasting increases in synaptic efficacy. LTP is probably also important in the generation of seizures since it is critical to the development of kindling (Racine *et al*, 1983; Vezzani *et al*, 1988). Selective NMDA antagonists such as MK-801 and D-2-amino-5-phosphonovalerate (AVP) have provided tools with which to explore the functions of this system. Blockade of NMDA receptors prevents kindling-like phenomena *in vitro* (Slater *et al*, 1985; Anderson *et al*, 1987) and *in vivo* (McNamara *et al*, 1988; Vezzani *et al*, 1988). To date there have been few studies looking at the effects of alcohol on NMDA function, although potentially such defects could explain the cognitive and neuropathological defects seen in chronic alcoholics (see Lishman, 1987). Acute doses of alcohol reduce NMDA function in that there is a reduction in NMDA-stimulated calcium (Ca) uptake and Ca-dependent production of cyclic guanosine monophosphate (Hoffman *et al*, 1989*b*). Sensitivity of hippocampal neurons to NMDA (Lovinger *et al*, 1989) and of Purkinje neurones to glutamate (Franklin & Gruol, 1987) is reduced. Although chronic alcohol reduces LTP in rat hippocampal neurones (Durand & Carlen, 1984), MK-801 binding is increased by approximately 25% in this area, perhaps in compensation. It appears that NMDA function is increased in alcohol withdrawal, as an increase in hippocampal MK-801 binding of up to 80% has been recently described (Hoffman *et al*, 1989*a*).

Calcium channels

Experiments have demonstrated an important role for Ca channels in the chronic effects of alcohol. Lynch & Littleton (1983) showed neurones were sensitised to Ca by chronic alcohol exposure. Later studies revealed an increase in the subtype of Ca channel labelled by the dihydropyridines (Dolin *et al*, 1987). Central effects of increased numbers of Ca channels would produce increased neuronal excitability, while peripheral changes may include increased adrenal catecholamine release (see Littleton, 1989). Blockade of these channels during alcohol withdrawal has been demonstrated to attenuate some symptoms, especially the seizures (Little *et al*, 1986; Littleton & Little, 1988). Administration of Ca antagonists before withdrawal, reduced tolerance development and withdrawal symptoms, and prevented the increase in dihydropyridine binding (Whittington & Little, 1989; Dolin & Little, 1990). Trials of Ca antagonists in alcohol withdrawal in animals (Little *et al*, 1986) have shown improvement in physical symptoms, especially seizures, although Bone *et al* (1989) suggested that benzodiazepines are more effective against other symptoms such as tremor. Preliminary work in humans suggests that Ca antagonists may be of some use clinically (Koppi *et al*, 1987). More comprehensive studies are clearly required in this area.

Excitatory interactions

Clearly, there are many interactive factors that may contribute to alcohol withdrawal. It would appear that a series of positive-feedback loops are generated, and these lead to massive central hyperexcitability (see Fig. 1). For instance, there are mutual interactions between adrenergic and CRF systems. CRF stimulates NA neurones of the locus coeruleus (Al-Damluji *et al*, 1987), the major source of ascending NA neurones in the CNS (Swanson, 1976). Direct injections of CRF into this area disrupts NA neuronal responses to sensory stimulation with the production of excessive and irregular firing patterns (Valentino *et al*, 1983). Additionally, administration (i.c.v.) of CRF stimulates sympathetic nervous system activity (Brown *et al*, 1982). Finally, CRF-conditioned fear responses are antagonised by the beta-blocker propranolol (Cole & Koob, 1988).

Corticotrophin-releasing factor neurones are themselves innervated by catecholaminergic neurones (Kitazawa *et al*, 1987), and NA can stimulate CRF release (Alonso *et al*, 1986). In this way, CRF stimulation of NA pathways could lead to a positive-feedback loop that results in greater CRF secretion (Fig. 1). Processes like this may underlie CRF kindling (Weiss *et al*, 1986). A further factor provoking CRF release may be the reduction of alpha-2-adrenoceptor inhibition of NA release. This has been demonstrated in alcohol withdrawal for other alpha-2-adrenoceptor-mediated responses (Nutt *et al*, 1988*b*).

Stimulation of NMDA receptors releases NA from hippocampal slices (Jones *et al*, 1987; Lalies *et al*, 1988). Overactivity of NMDA receptors due to low Mg would accentuate NA release, which may in turn further reduce Mg levels, leading to further increases in NMDA (see Fig. 1). Since DA release from nigral slices is also potentiated by excitatory amino acids via an NMDA receptor and can be inhibited by Mg (Araneda & Bustos, 1989), NMDA receptor overactivity may produce excessive DA release.

Receptor dysfunction and clinical symptoms

There are many symptoms and signs in alcohol withdrawal but these fall into a few clusters. Three areas can be determined in relation to the receptor disturbances described above (Table I). These are: increased sympathetic activity, kindling and seizures, and hallucinations and DTs. The increase in sympathetic activity produces symptoms such as tremor, sweating and anxiety, the severity of which have been found to be correlated with indices of central noradrenergic overactivity (Hawley *et al*, 1985). These symptoms respond to administration of the alpha-2-adrenoceptor agonists clonidine (Wilkins *et al*, 1983; Glue & Nutt, 1987) and lofexidine (Brunning *et al*, 1986), which act to reduce sympathetic outflow (Kobinger, 1984). However, it should be noted that clonidine appears to be ineffective in stopping more severe withdrawal symptoms and seizures (Robinson *et al*, 1989), and benzodiazepines are still the best treatment for the global management of alcohol withdrawal.

TABLE I

Summary of possible relationships of neurotransmitter disturbances to symptoms of alcohol withdrawal (arrows indicate inhibition (↓) or excitation (↑) of neurotransmitter)

Symptoms	*Neurotransmitter disturbances*
Increased sympathetic activity	↑NA; ↑CRF; ↓GABA
Hallucinations/DTs	↑DA
Seizures kindling	↓GABA; ↑NMDA (↓Mg)[1]; ↑CRF

1. ↑NMDA related to ↓Mg.

Kindling has been used to explain a number of clinical features of alcoholism and alcohol withdrawal, especially the progressive worsening of symptoms and signs with prolonged drinking, and increased risk of seizures (Ballenger & Post, 1978; Brown *et al*, 1988). Despite this being a compelling hypothesis there is little evidence to support it (Ballenger & Post, 1978). The only pharmacological evidence from humans is that indices of NA activity are correlated with duration of drinking (Nutt *et al*, 1988*b*). Further support for this hypothesis comes from the probable stimulatory effects of alcohol administration and withdrawal upon CRF and NMDA activity, and their roles in producing kindling.

During alcohol withdrawal, symptoms occur at particular times after stopping alcohol rather than all at once or randomly (Victor & Adams, 1953; Kanzow, 1986). For instance, symptoms associated with increased sympathetic drive (such as anxiety, tremor and sweating) tend to be maximal 24 hours after stopping drinking, whereas seizures occur 24–48 hours after the last drink. The severe agitation and delirium, vivid hallucinations and autonomic overactivity of DTs occur between 48–72 hours after the cessation of drinking (Victor & Adams, 1953; Victor, 1966). The role of NA overactivity in early withdrawal symptoms of tremor and sweating has been clearly demonstrated (Hawley *et al*, 1985). We hypothesise that there is overactivity of NMDA and CRF systems which may contribute to these early signs and also lead to the occurrence of seizures.

The receptor disturbances underlying DTs are more speculative, but may well involve DA overactivity as well as kindling. Alcohol withdrawal possibly has an initiating effect, with the DTs then occurring as a result of short-term kindling (as described above with CRF). The DTs differ from other withdrawal phenomena in that they are difficult to bring under control using presently available treatment regimes (Thompson *et al*, 1975; Kramp *et al*, 1979). Hypnosedative drugs are more effective in reducing the incidence of DTs than DA-antagonists such as haloperidol (Palsson, 1986). However, once DTs have developed, these drugs influence different groups of symptoms. For instance, the delirium of DTs has been shown to respond to haloperidol, which has little effect on autonomic symptoms (Holzbach & Buhler, 1978). It would be interesting to know if NMDA antagonists are of use in treating this state. One approach to this might be to use Mg supplements, since other NMDA antagonists are not yet available for clinical use.

Therapeutic possibilities

Present pharmacological practice for alcohol withdrawal is to use one of the hypnosedative drugs, chlormethiazole or benzodiazepines. The use of loading-dose benzodiazepines has been demonstrated to be clinically efficacious, very cost- and time-effective, and probably the preferred treatment for withdrawal at present (Sellers *et al*, 1983). The shorter half-life and lower therapeutic ratio of chlormethiazole makes it a less attractive treatment. Supplementation of Mg, although it may be included in concomitant vitamin and nutritional supplementation, is less frequently used, and may have a considerably greater role due to its action on NMDA receptors. Clonidine, an alpha-2-adrenoceptor agonist, which reduces NA release, has been used as a treatment for alcohol withdrawal (Bjorkqvist, 1975; Walinder *et al*, 1981), although it is only of limited benefit in severe withdrawal (Robinson *et al*, 1989), and should not be used as sole treatment in patients with a history of seizures. In combination with hypnosedatives, neuroleptics have a place in the management of DTs, with drugs such as haloperidol being preferred to the less dopamine-selective compounds (e.g. phenothiazines) as the latter are associated with increased incidence of seizures and DTs (Thomas & Freedman, 1965; Golbert *et al*, 1967; Kaim *et al*, 1969; Feuerlein & Reiser, 1986). Potentially, newer DA antagonists of markedly greater receptor selectivity (e.g. pimozide) may prove to be more efficacious, since there is some evidence to suggest a reduced tendency to potentiate seizures in animals (Meldrum *et al*, 1975). Finally, Ca antagonists given either before or during alcohol withdrawal are promising therapeutic agents, although further clinical trials are required in this area.

One aspect of withdrawal treatment that is little recognised is kindling. This is suggested by the tendency for withdrawal symptoms to recur more rapidly and to increase in severity with repeated episodes of drinking (Ballenger & Post, 1978). Given the considerable evidence from animal models of the role of NMDA and CRF in kindling, and their increased activity during chronic alcohol intake and withdrawal, specific treatments to slow down or stop kindling processes may be of great importance. In view of the dangers of kindling, Linnoila (1987) suggested that withdrawal symptoms should receive aggressive medical treatment, to prevent the progression of kindling phenomena. This challenges the notion that withdrawal is a relatively self-limiting and harmless event that may be managed without medical intervention. It suggests that such a policy might increase the risk of kindling, and lead to long-term problems of exacerbated withdrawals and their many sequelae. In other words, there is now good evidence that the state of increased excitability of the brain accompanying withdrawal may cause long-lasting changes. These are manifest as worsening (kindling) of withdrawal symptoms in subsequent drinking bouts. Active management of the earlier withdrawals could be expected to prevent this.

One important consequence not yet discussed is alcoholic dementia. There is good evidence that NMDA receptor overactivity (Olney, 1984) and hypercortisolaemia (Sapolsky & Pulsinelli, 1985) lead to hippocampal cell death. The long-term cognitive dysfunction of alcoholic dementia could be produced by loss of these neurones (Walker *et al*, 1980). The potential of selective NMDA and CRF antagonists to prevent this process could be of great clinical benefit. As mentioned above, they might also be of value in treating DTs.

The NMDA antagonists that are presently available are anticonvulsant and neuroprotective in animal models (Croucher *et al*, 1982; Schwarcz *et al*, 1982; Schwarcz & Meldrum, 1985). However, as related compounds such as phencyclidine cause hallucinations in humans (Snyder *et al*, 1980), this may limit the usefulness of NMDA antagonists. It would seem that Mg supplementation is the only viable way at present of reducing NMDA receptor activity in humans. CRF antagonists are only just becoming available, and being large peptides, would be unlikely to be peripherally active. The discovery of a stable non-peptide analogue is awaited with interest.

Acknowledgements

We are indebted to Drs Graham Collingridge and Hilary Little for their helpful comments.

References

Adinoff, B. (1987) Hypothalamic-pituitary-adrenal axis function in recently abstinent alcoholics. In *Alcohol Withdrawal and Noradrenergic Function* (Moderator: M. Linnoila). *Annals of Internal Medicine*, **107**, 875–889.

Al-Damluji, S., Perry, L., Tomlin, S., *et al* (1987) Alpha-adrenergic stimulation of corticotropin secretion by a specific central mechanism in man. *Neuroendocrinology*, **45**, 68–76.

Allan, A. M. & Harris, R. A. (1987) Involvement of neuronal chloride channels in ethanol intoxication, tolerance and dependence. In *Recent Developments in Alcoholism, Vol. 5* (ed. M. Galanter), pp. 313–325. New York: Plenum Press.

Alonso, G., Szafarczyk, A., Balmefrezol, M., *et al* (1986) Immunocytochemical evidence for stimulatory control by the ventral noradrenergic bundle of parvocellular neurons of the paraventricular nucleus secreting corticotropin releasing hormone and vasopressin in rats. *Brain Research*, **397**, 297–307

Anderson, W. W., Swartzwelder, H. S. & Wilson, W. A. (1987) The NMDA antagonist 2-amino-5-phosphonovalerate blocks stimulus train-induced epileptogenesis but not epileptiform bursting in the rat hippocampal slice. *Journal of Neurophysiology*, **57**, 1–21.

Annunziato, L., Amoroso, S., Di Renzo, G., *et al* (1983) Increased GH responsiveness to dopamine receptor stimulation in alcohol addicts during the late withdrawal syndrome *Life Sciences*, **33**, 2651–2655.

Araneda, R. & Bustos, G. (1989) Modulation of dendritic release of dopamine by N-methyl-aspartate receptors in rat substantia nigra. *Journal of Neurochemistry*, **52**, 962–970

Ballenger, J. C. & Post, R. M. (1978) Kindling as a model for alcohol withdrawal syndromes. *British Journal of Psychiatry*, **133**, 1–14.

Banna, N. R. (1969) Potentiation of cutaneous inhibition by ethanol. *Experientia*, **25**, 619–620.

Bjorkqvist, S. E. (1975) Clonidine in alcohol withdrawal. *Acta Psychiatrica Scandinavica*, **52**, 256–263

Bone, G. H. A., Majchrowicz, E., Martin, P. R., *et al* (1989) A comparison of calcium antagonists and diazepam in reducing alcohol withdrawal tremors. *Psychopharmacology*, **99**, 386–388.

Borg, S., Kvande, H. & Sedvall, G. (1981) Central norepinephrine metabolism during alcohol intoxication in addicts and healthy volunteers. *Science*, **213**, 1135–1137.

Brown, M. E., Anton, R. F., Malcolm, R., *et al* (1988) Alcohol detoxification and withdrawal seizures: clinical support for a kindling hypothesis. *Biological Psychiatry*, **23**, 507–514.

Brown, M. R., Fisher, L. A., Rivier, J., *et al* (1982) Corticotropin-releasing factor: effects on the sympathetic nervous system and oxygen consumption. *Life Sciences*, **30**, 207–210.

Brunning, J., Mumford, J. P. & Keaney, F. P. (1986) Lofexidine in alcohol withdrawal states. *Alcohol and Alcoholism*, **21**, 167–170.

Coffman, J. A. & Petty, F. (1985) Plasma GABA levels in chronic alcoholics. *American Journal of Psychiatry*, **142**, 1204–1205.

Cole, B. J. & Koob, G. F. (1988) Propranolol antagonises the enhanced conditioned fear produced by corticotropin releasing factor. *Journal of Pharmacology and Experimental Therapeutics*, **247**, 902–910.

Collingridge, G. L. & Bliss, T. V. P. (1987) NMDA receptors – their role in long-term potentiation. *Trends in Neurological Science*, **10**, 288–293.

Cott, J. A., Carlsson, A., Engel, J., *et al* (1976) Suppression of ethanol-induced locomotor stimulation by GABA-like drugs. *Naunyn-Schmiedeberg's Archives of Pharmacology*, **295**, 203–209.

Cowen, P. J. & Nutt, D. J. (1982) Abstinence syndromes after withdrawal of tranquilising drugs: a common neurochemical mechanism? *Lancet*, *ii*, 360–362.

Croucher, M. J., Collins, J. F. & Meldrum, B. S. (1982) Anticonvulsant action of excitatory amino acid antagonists. *Science*, **216**, 899–901.

Daus, A. T., Freeman, W. M., Wilson, J., *et al* (1985) Clinical experience with 781 cases of alcoholism evaluated and treated on an inpatient basis by various methods. *International Journal of Addiction*, **20**, 643–650.

Dave, J. R., Eiden, L. E., Karanian, J. W., *et al* (1986) Ethanol exposure decreases pituitary corticotropin-releasing factor binding, adenylate cyclase activity, proopiomelanocortin biosynthesis, and plasma beta-endorphin levels in the rat. *Endocrinology*, **118**, 280–286.

Davidoff, R. S. (1973) Alcohol and presynaptic inhibition in isolated spinal cord preparation. *Archives of Neurology*, **28**, 60–63.

Dolin, S. I. & Little, H. J. (1990) Calcium antagonists during ethanol administration. *Journal of Pharmacology and Experimental Therapeutics* (in press).

——, ——, Hudspith, M., *et al* (1987) Increased dihydropyridine-sensitive calcium channels in rat brain may underlie ethanol physical dependence. *Neuropharmacology*, **26**, 275–279

Durand, D. & Carlen, P. L. (1984) Impairment of long-term potentiation in rat hippocampus following chronic ethanol treatment. *Brain Research*, **308**, 325–332.

Ehlers, C. L. & Chaplin, R. I. (1987) Chronic ethanol exposure potentiates the locomotor-activating effects of corticotropin-releasing factor (CRF) in rats. *Regulatory Peptides*, **19**, 345–353.

——, Henriksen, S., Wang, M., *et al* (1983) Corticotropin-releasing factor increases brain excitability and convulsive seizures in the rat. *Brain Research*, **278**, 332–336.

Engel, J. & Liljeqvist, S. (1976) The effect of long-term ethanol treatment on the sensitivity of dopamine receptors in the nucleus accumbens. *Psychopharmacology*, **49**, 253–257.

Fadda, F., Mosca, E., Meloni, R., *et al* (1985) Suppression by progabide of ethanol withdrawal syndrome in rats. *European Journal of Pharmacology*, **109**, 321–325.

Feuerlein, W. & Reiser, E. (1986) Parameters affecting the course and results of delirium tremens treatment. *Acta Psychiatrica Scandinavica* (suppl. 329), 120–123.

Flink, E. B. (1986) Magnesium deficiency in alcoholism. *Alcoholism: Clinical and Experimental Research*, **10**, 590–594.

——, McCollister, R., Prasad, A. S., *et al* (1957) Evidence for clinical magnesium deficiency. *Annals of Internal Medicine*, **47**, 956–968.

Franklin, C. L. & Gruol, D. L. (1987) Acute ethanol alters the firing pattern and glutamate response of cerebellar Purkinje neurons in culture. *Brain Research*, **416**, 205–218.

Freund, G (1980) Benzodiazepine receptor loss in brains of mice after chronic alcohol consumption. *Life Sciences*, **27**, 987–992.

Frye, G. D. & Breese, G. R. (1982) GABAergic modulation of ethanol-induced motor impairment. *Journal of Pharmacology and Experimental Therapeutics*, **223**, 750–756.

Gallimberti, L., Canton, G , Gentile, N., *et al* (1989) Gamma-hydroxybutyric acid for treatment of alcohol withdrawal syndrome. *Lancet*, *ii*, 787–789.

Garthwaite, G. & Garthwaite, J. (1986) Amino acid toxicity: intracellular sites of calcium accumulation associated with the onset of irreversible damage to rat cerebellar neurones in vitro. *Neuroscience Letters*, **71**, 53–58.

Glue, P. & Nutt, D J. (1987) Clonidine in alcohol withdrawal: a pilot study of differential symptom responses following i.v. clonidine. *Alcohol and Alcoholism*, **22**, 161–166

——, Sellman, J. D., Nicholl, M G., *et al* (1989) Studies of alpha-2-adrenoceptor function in abstinent alcoholics. *British Journal of Addiction*, **84**, 97–102

Golbert, T. M., Sanz, C. J., Rose, H. D , *et al* (1967) Comparative evaluation of treatments of alcohol withdrawal syndromes *Journal of the American Medical Association*, **201**, 113–116.

Hawley, R. J., Major, L. F., Schulman, E., *et al* (1981) Cerebrospinal fluid cyclic nucleotides and GABA do not change in alcohol withdrawal. *Life Sciences*, **28**, 295–299.

——, Major, L. F., Schulman, E., *et al* (1985) Cerebrospinal fluid 3-methoxy-4-hydroxyphenylglycol and norepinephrine levels in alcohol withdrawal. *Archives of General Psychiatry*, **42**, 1056–1062.

Heaton, F. W., Pyrah, L. N., Beresford, C. C., *et al* (1962) Hypomagnesaemia in chronic alcoholism *Lancet*, *ii*, 802–805.

Herron, C. E., Lester, R A J., Coan, E J., *et al* (1985*a*) Intracellular demonstration of an N-methyl-D-aspartate receptor mediated component of synaptic transmission in the rat hippocampus. *Neuroscience Letters*, **60**, 19–23.

——, Williamson, R. & Collingridge, G. L. (1985*b*) A selective N-methyl-D-aspartate antagonist depresses epileptiform activity in rat hippocampal slices. *Neuroscience Letters*, **61**, 255–260.

Heuser, I., Von Bardeleben, U., Boll, E., *et al* (1988) Response of ACTH and cortisol to human corticotropin-releasing hormone after short-term abstention from alcohol abuse. *Biological Psychiatry*, **24**, 316–321.

Hoffman, P. L., Moses, F., Hudspith, M., *et al* (1989*a*) Ethanol: potent and selective inhibitor of NMDA-stimulated cyclic GMP production. *Research Society on Alcohol Abstracts, Colorado*, **312** (abstr. 43).

——, Rabe, C. S., Moses, F., *et al* (1989*b*) N-methyl-D-aspartate receptors and ethanol: inhibition of calcium flux and cyclic GMP production. *Journal of Neurochemistry*, **52**, 1937–1940.

Holsboer, F , Von Bardeleben, U., Buller, R., *et al* (1987) Stimulation response to corticotropin-releasing hormone (CRH) in patients with depression, alcoholism and panic disorder. *Hormonal and Metabolic Research* (suppl. 16), 80–88.

Holzbach, E. & Buhler, K. E. (1978) Die behandlung des delirium tremens mit haldol. *Nervenartz*, **49**, 405–409

Jones, S. M., Snell, L. D. & Johnson, K. M (1987) Phencyclidine selectively inhibits N-methyl-D-aspartate-induced hippocampal (3H)norepinephrine release. *Journal of Pharmacology and Experimental Therapeutics*, **240**, 492–497

Kaim, S. C., Klett, C. J. & Rothfield, B. (1969) Treatment of the acute alcohol withdrawal syndrome: a comparison of four drugs. *American Journal of Psychiatry*, **125**, 1640–1646.

Kanzow, W. T (1986) The clinical stages of alcoholic delirium and their therapeutic significance. *Acta Psychiatrica Scandinavica* (suppl. 329), 124–128.

Kitazawa, S., Shioda, S & Nakai, Y. (1987) Catecholaminergic innervation of neurons containing corticotropin-releasing factor in the paraventricular nucleus of the rat hypothalamus. *Acta Anatomica*, **129**, 337–343.

Kobinger, W. (1984) Alpha-2-adrenoceptors in cardiovascular regulation of norepinephrine In *Frontiers of Clinical Science, Vol. 2* (eds M. G. Ziegler & C. R. Lake), pp. 307–326. Baltimore: Williams and Wilkins.

Koob, G. F. & Bloom, F. E (1985) Corticotropin-releasing factor and behaviour. *Federation Proceedings*, **44**, 259–263.

Koppi, S., Eberhardt, G., Haller, R., *et al* (1987) Calcium channel blocking agent in the treatment of acute alcohol withdrawal – caroverine versus meprobamate in a randomised double blind study. *Neuropsychobiology*, **17**, 49–52.

Kramp, P., Ronsted, P. & Hansen, T. (1979) Barbital and diazepam plasma levels during treatment of delirium tremens. *Acta Psychiatrica Scandinavica*, **59**, 263–275.

Krystal, H. (1959) The physiological basis of the treatment of delirium tremens. *American Journal of Psychiatry*, **116**, 137–147

Lalies, M., Middlemiss, D. N. & Ransom, R. (1988) Stereoselective antagonism of NMDA-stimulated noradrenaline release from rat hippocampal slices by MK-801. *Neuroscience Letters*, **91**, 339–342.

Liljeqvist, S. (1978) Changes in the sensitivity of dopamine receptors in the nucleus accumbens and in the striatum induced by chronic ethanol administration. *Acta Pharmacologica et Toxicologica*, **43**, 19–28.

—— & Engel, J. (1982) Effects of GABAergic agonists and antagonists on various ethanol-induced behavioural changes. *Psychopharmacology*, **78**, 71–75.

Linnoila, M. (1987) Alcohol withdrawal and noradrenergic function. *Annals of Internal Medicine*, **107**, 875–889.

Lishman, W. A (1987) Brain damage in alcoholism: current concepts. *Acta Medica Scandinavica* (suppl. 717), 5–17.

Lister, R. G. & Karanian, J. W. (1987) RO 15-4513 induces seizures in DBA/2 mice undergoing alcohol withdrawal. *Alcohol*, **4**, 409–411

—— & Nutt, D. J. (1987) Is RO 15-4513 a specific alcohol antagonist? *Trends in Neurological Science*, **10**, 223–225.

Little, H. J , Dolin, S. J. & Halsey, M. J. (1986) Calcium channel antagonists decrease the ethanol withdrawal syndrome in rats. *Life Sciences*, **39**, 2059–2065

Littleton, J. (1989) Alcohol intoxication and physical dependence: a molecular mystery tour. *British Journal of Addiction*, **84**, 267–276.

—— & Little, H. J. (1988) Dihydropyridine-sensitive Ca channels in brain are involved in the central nervous system hyperexcitability associated with alcohol withdrawal states. *Annals of the New York Academy of Science*, **552**, 199–202.

Lovinger, D. M., White, G. & Weight, F. F. (1989) Ethanol inhibits NMDA-activated ion current in hippocampal neurons. *Science*, **243**, 1721–1724.

Lynch, M. A. & Littleton, J. M. (1983) Possible association of alcohol tolerance with increased synaptic Ca sensitivity. *Nature*, **303**, 175–177.

Marley, R. J. & Wehner, J. M. (1987) GABA enhancement of flunitrazepam binding in mice selectively bred for differential sensitivity to ethanol. *Alcohol and Drug Research*, **7**, 25–32.

Martz, A., Deitrich, R. A. & Harris, R. A. (1983) Behavioural evidence for the involvement of gamma-aminobutyric acid in the actions of ethanol. *European Journal of Pharmacology*, **89**, 53–62.

Meldrum, B., Anelzark, G. & Trimble, M. (1975) Drugs modifying dopaminergic activity and behaviour, the EEG, and epilepsy in *papio papio*. *European Journal of Pharmacology*, **32**, 203–213.

McNamara, J. O., Russell, R. D., Rigsbee, L., *et al* (1988) Anticonvulsant and antiepileptogenic actions of MK-801 in the kindling and electroshock models. *Neuropharmacology*, **27**, 563–568.

Nesteros, J. M. (1980) Ethanol specifically potentiates GABA-mediated neurotransmission in feline cerebral cortex. *Science*, **209**, 708–710.

Nowak, L., Bregestovski, P., Ascher, P., *et al* (1984) Magnesium gates glutamate-activated channels in mouse central neurones. *Nature*, **307**, 462–465.

Nutt, D. J. & Glue, P. (1986) Monoamines and alcohol. *British Journal of Addiction*, **81**, 327–338.

——, Adinoff, B., Ravitz, B., *et al* (1988*a*) Cerebro-spinal fluid studies in alcoholics and violent offenders. *Australian Drug and Alcohol Review*, **7**, 105–108.

——, Glue, P., Molyneux, S., *et al* (1988*b*) Alpha-2-adrenoceptor activity in alcohol withdrawal: a pilot study of the effects of i.v. clonidine in alcoholics and normals. *Alcoholism: Clinical and Experimental Research*, **12**, 14–18.

Olney, J. W. (1984) Excitotoxins: an overview. In *Excitotoxins* (eds K. Fuxe, P. J. Roberts & R. Schwarcz), p. 82–96. London: Macmillan.

Palsson, A. (1986) The efficacy of early chlormethiazole medication in the prevention of delirium tremens. A retrospective study of the outcome of different treatment strategies at the Helsingborg psychiatric clinics, 1975–1980. *Acta Psychiatrica Scandinavica* (suppl 329), 140–145.

Potter, J. F., Bannan, L. T. & Beevers, D. G. (1984) The effect of a non-selective lipophilic beta-blocker on the blood pressure and noradrenaline, vasopressin, cortisol and renin release during alcohol withdrawal. *Clinical Experiments in Hypertension – Theory and Practice*, **A6**, 1147–1160.

Racine, R. J., Milgram, N. W. & Hafner, S. (1983) Long-term potentiation phenomena in the rat limbic forebrain. *Brain Research*, **260**, 217–231.

Rastogi, S. K., Thyagarajan, R., Clothier, J., *et al* (1986) The effect of chronic treatment of ethanol on benzodiazepine and picrotoxin sites on the GABA-receptor complex in regions of the brain of the rat. *Neuropharmacology*, **25**, 1179–1184.

Risher-Flowers, D., Adinoff, B., Ravitz, B., *et al* (1988) Circadian rhythms of cortisol during alcohol withdrawal. *Advances in Alcohol and Substance Abuse*, **7**, 37–41.

Rivier, C., Bruhn, T. & Vale, W. (1984) Effect of ethanol on the hypothalamic-pituitary-adrenal axis in the rat: role of corticotropin-releasing factor (CRF). *Journal of Pharmacology and Experimental Therapeutics*, **229**, 127–131.

Robinson, B. J., Robinson, G. M., Maling, T. J. B., *et al* (1989) Is clonidine useful in the treatment of alcohol withdrawal? *Alcoholism: Clinical and Experimental Research*, **13**, 95–98.

Rottenberg, H. (1985) Alcohol modulation of benzodiazepine receptors. *Alcohol*, **2**, 203–207.

Sapolsky, R. M. & Pulsinelli, W. A. (1985) Glucocorticoids potentiate ischaemic injury to neurons: therapeutic implications. *Science*, **229**, 1397–1400.

Shilicovich, A., Duvilanski, B., Gonzales, N. N., *et al* (1985) The effect of acute ethanol administration on GABA receptor binding in cerebellum and hypothalamus. *European Journal of Pharmacology*, **111**, 365–369.

Sellers, E. M., Naranjo, C. A., Harrison, M., *et al* (1983) Diazepam loading: simplified treatment of alcohol withdrawal. *Clinical Pharmacology and Therapeutics*, **34**, 822–826.

Schwarcz, R. & Meldrum, B. (1985) Excitatory aminoacid antagonists provide a therapeutic approach to neurological disorders. *Lancet*, *ii*, 140–143.

——, Collins, J. F. & Parks, D. A. (1982) α-Aminophosphonocarboxylates block ibotenate but not kainate neurotoxicity in rat hippocampus. *Neuroscience Letters*, **33**, 85–90.

Slater, N. T., Stelzer, A. & Galvan, M. (1985) Kindling-like stimulus patterns induce epileptiform discharges in the guinea pig in vitro hippocampus. *Neuroscience Letters*, **60**, 25–31.

Snyder, S. H. (1980) Phencyclidine. *Nature*, **285**, 355–356.

Stelzer, A., Slater, N. T. & Bruggencate, G. (1987) Activation of NMDA receptors blocks GABAergic inhibition in an in vitro model of epilepsy. *Nature*, **326**, 698–701.

Suzdak, P. D., Schwartz, R. D., Skolnick, P., *et al* (1986*a*) Ethanol stimulates gamma-aminobutyric acid receptor-mediated chloride transport in rat brain synaptoneurosomes. *Proceedings of the National Academy of Sciences*, **83**, 4071–4075.

——, Glowa, J. R., Crawley, J. N., *et al* (1986*b*) A selective imidazobenzodiazepine antagonist of ethanol in the rat. *Science*, **234**, 1243–1247.

Swanson, L. W. (1976) The locus coeruleus: a cytoarchitectonic, golgi and immunohistochemical study in the albino rat. *Brain Research*, **110**, 39–56.

Taylor, K. M. & Laverty, R. (1969) The effect of chlordiazepoxide, diazepam and nitrazepam on catecholamine metabolism in regions of the rat brain. *European Journal of Pharmacology*, **8**, 296–301.

Thomas, D. W. & Freedman, D. X. (1964) Treatment of the alcohol withdrawal syndrome. *Journal of the American Medical Association*, **188**, 316–318.

Thompson, W. L., Johnson, A. D. & Maddrey, W. L. (1975) Diazepam and paraldehyde for treatment of severe delirium tremens. *Annals of Internal Medicine*, **82**, 175–180.

Ticku, M. K. & Davis, W. C. (1981) Evidence that ethanol and pentobarbital enhance 3H-diazepam binding at the benzodiazepine-GABA receptor-ionophore complex indirectly. *European Journal of Pharmacology*, **71**, 521–522.

—— & Rastogi, S. K. (1986) Barbiturate-sensitive sites on the benzodiazepine-GABA receptor-ionophore complex. In *Molecular and Cellular Mechanisms of Anaesthetics* (eds S. H. Roth & K. W. Miller), pp. 178–188. New York: Plenum Press.

——, Burch, T. P. & Davis, W. C. (1983) The interactions of ethanol with the benzodiazepine-GABA receptor-ionophore complex. *Pharmacology Biochemistry and Behaviour*, **18**, 15–18.

——, Lowrimore, P. & Lehoullier, P. (1986) Ethanol enhances GABA-induced 36-Cl-influx in primary spinal cord cultured neurons. *Brain Research Bulletin*, **17**, 123–126.

Tran, V. T., Snyder, S. H., Major, L. F., *et al* (1981) GABA receptors are increased in the brains of alcoholics. *Annals of Neurology*, **9**, 289–292.

Vale, W., Speiss, J., Rivier, C., *et al* (1981) Characterization of a 41-residue ovine hypothalamic peptide that stimulates secretion of corticotropin and beta-endorphin. *Science*, 213, 1394–1397.

Valentino, R. J., Foote, S. L. & Aston-Jones, G. (1983) Corticotropin-releasing factor activates noradrenergic neurons of the locus coeruleus. *Brain Research*, **270**, 363–367.

Valimaki, M., Pelkonen, R., Harkonen, M., *et al* (1984) Hormonal changes in noncirrhotic male alcoholics during ethanol withdrawal. *Alcohol and Alcoholism*, **19**, 235–242.

VEZZANI, A., WU, H-Q., MONTENA, E., *et al* (1988) Role of the N-methyl-D-aspartate-type receptors in the development and maintenance of hippocampal kindling in rats *Neuroscience Letters*, **87**, 63–68

VICTOR, M. (1966) Treatment of alcoholic intoxication and the withdrawal syndrome. *Psychosomatic Medicine*, **28**, 636–650.

—— & ADAMS, R. D. (1953) The effects of alcohol on the nervous system. *Research Publications of the Association for Nervous and Mental Diseases*, **32**, 526–573.

WALINDER, J., BALLDIN, J., BOKSTROM, K., *et al* (1981) Clonidine suppression of the alcohol withdrawal syndrome. *Drug and Alcohol Dependence*, **8**, 345–348.

WALKER, D. W., BARNES, D. E., ZORNETZER, S. F., *et al* (1980) Neuronal loss in hippocampus induced by prolonged ethanol consumption in rats. *Science*, **209**, 711–713

WATKINS, J. C. & EVANS, R. H. (1981) Excitatory amino acid transmitters. *Annual Reviews in Pharmacology and Toxicology*, **21**, 165–204.

WEISS, S. R. B., POST, R. M, GOLD, P. W., *et al* (1986) CRF-induced seizures and behaviour: interaction with amygdala kindling. *Brain Research*, **372**, 345–351.

WHITTINGTON, M. A. & LITTLE, H. J. (1988) Nitrendipine prevents the ethanol withdrawal syndrome when administered chronically with ethanol prior to withdrawal. *British Journal of Pharmacology*, **94**, 385.

WHYTE, K. F., ADDIS, G. J., WHITESMITH, R., *et al* (1987) Adrenergic control of plasma magnesium in man. *Clinical Science*, **72**, 135–138.

WILKINS, A. J., JENKINS, W. J & STEINER, J. A. (1983) Efficacy of clonidine in the treatment of the alcohol withdrawal syndrome. *Psychopharmacology*, **81**, 78–80.

WOLFE, S. M. & VICTOR, M (1969) The relationship of hypomagnesaemia and alkalosis to alcohol withdrawal symptoms. *Annals of the New York Academy of Science*, **162**, 973–984

*Paul Glue, MRCPsych, *Reckitt and Colman Psychopharmacology Unit, Department of Pharmacology, The Medical School, University Walk, Bristol BS8 1TD*; David Nutt, DM, MRCP, MRCPsych, *Reckitt and Colman Psychopharmacology Unit, Bristol*

*Correspondence

British Journal of Psychiatry (1990), **157**, 500–514

Towards a System of Outcome Indicators for Mental Health Care

RACHEL JENKINS

A system of outcome indicators for mental health care is urgently needed in order to ensure that clinicians, district health authorities, and directors of public health can monitor and evaluate mental health care. Theoretical aspects of health care indicators and the various classes of outcome measures available can be used to draw up a preliminary system of indicators of health care input, process and outcome for the major categories of mental illness, including schizophrenia, affective psychosis, neurosis, dementia, mental handicap, child psychiatry, forensic psychiatry, alcohol and drugs. Such a system is not intended to be definitive or exhaustive but rather to form a basis for development by clinicians, researchers and planners for their own requirements.

The requirement for mental health care outcome indicators

In order to evaluate our health-care system, we need to be able to measure the baseline health of the population, and then to measure the impact of health care on that baseline. We need to be able to monitor and evaluate progress towards more effective health care and better health, to evaluate the efficacy of health-promotion and illness-prevention programmes, and to improve resource allocation in health care. In order to do this effectively in a valid and reproducible manner, health indicators are required. Besides the more global indicators of general health, lifestyles, quality of life, and health equity, we also need indicators relevant to the different categories of illness, and to specific strategies to prevent diseases, to alleviate disability, and to restore function.

While the call for such indicators has hitherto come primarily from planners and policy makers, it should be abundantly clear that indicators are also needed by clinicians if they are in any way to evaluate the effectiveness of their work and to identify areas for improvement, and if district and regional health authorities and organisations such as the Hospital Advisory Service are to monitor quality of service. Particularly urgent requirements at the present time relate to district health authorities (DHAs) and directors of public health. In the 1989 White Paper "Working for Patients" (para 2: 11), DHAs are asked to:

> "concentrate on ensuring that the health needs of the population for which they are responsible are met; that there are effective services for the prevention and control of diseases and the promotion of health; that their population has access to a comprehensive range of high quality, value for money services; and on setting targets for and monitoring the performance of those management units for which they continue to have responsibility."

Thus DHAs in fulfilling their 'purchaser role' will need to develop the ability to monitor service provision, prevention programmes and quality of care, including health outcome.

There is a danger that measurement will concentrate on those features that are easily quantified rather than those that relate most directly to quality and health outcome. This danger exists in all clinical specialties, and mental health care is no exception. The ability to define and measure quality in mental health services is not well developed. Those aspects of service provision that can most easily be measured at present tend to be those which relate to service input and resources consumed rather than service outcome:

> "Not surprisingly, most existing mechanisms for external evaluation, such as registration of nursing homes, recognition of training posts, performance indicators, the Health Advisory Service and overseas health service accreditation have focused on resources and process rather than on outcomes." (Shaw, 1989)

The DHAs will need to construct a population profile that allows health status to be examined over time. In doing this, they will need to draw upon the epidemiological expertise of the directors of public health (see below). Each DHA needs to be able to monitor the quality of services provided by different providers in its area, to measure its progress against other DHAs in the region and nationally, to evaluate new service developments to ensure that objectives are being met, and to encourage each provider to be self-monitoring.

Following the Acheson report (Department of Health, 1988) on the future development of the public health function, the Department of Health issued new guidance to health authorities including the requirement that each district and region should appoint a director of public health (DPH) who must produce each year a report on the health of the population. The DPH is the successor to the medical officer of health, who was removed in the 1974 reorganisation.

The DPH's annual report is an independent assessment of the health of the resident population of the health district or region. The intention is to enable the district or region to discharge its responsibilities in relation to the health of its population. The report should present and interpret epidemiological evidence, identify local health problems including unmet need, and evaluate the outcome of existing services. Its recommendations for action should form an important input to the planning process. The long-term aim should be to reorientate the allocation of resources by health authorities to deal with the identified public health problems.

Indicators as measures

An 'indicator' is defined as a measure that summarises information relevant to a particular phenomenon, or a reasonable proxy for such a measure. Ideally, indicators should actually measure what they are supposed to measure (i.e. have validity), and they should provide the same answer if measured by different people in similar circumstances (i.e. have reliability) (Cook & Campbell, 1979). They should be able to measure change (i.e. have sensitivity), and they should reflect changes only in the situation concerned (i.e. have specificity) (World Health Organization, 1981). Of course in real life these criteria are hard to achieve, and indicators at best are indirect or partial measures of a complex situation. A *health* indicator, therefore, is a variable, susceptible to direct measurement, that reflects aspects of the state of health of a community, while a *health care* indicator is a variable that reflects aspects of the state of health care in a community (World Health Organization, 1981). Health care indicators can be categorised, using a general systems approach, into input, process, and outcome. The resources put into the health care system in terms of finance, personnel, building, etc., are the input, the activity of the personnel form the process, and the changes in functioning, morbidity and mortality are the outcome. (Thus, in general, outcome indicators in health care will be health indicators.) Outcome may of course be influenced not only by the health care system, but also by other aspects of public policy, social change, and environmental factors.

The measurement of input is relatively straightforward, a simple accounting procedure, and the measurement of process has been tackled for some time, in the shape of 'performance' or 'activity' indicators, although this has sometimes tended to follow the approach of establishing what is collectable first and seeing what it can tell us, rather than specifying the key aspects of performance and devising appropriate indicators.

The measurement of outcome is a more complex task, and the fact that outcome information is hard to obtain has led to health service inputs and processes being used as proxy measures for outcomes, which of course they are not (Bergner *et al*, 1979). It was also argued that because chronic health problems, morbidity and mortality are insensitive variables, and are seemingly unrelated to health care changes in the short term, the use of input and process indicators as proxy measures for outcome was justified. The initial assumption was that use is cure, and amount of use equates with severity of condition, but it is now well recognised that use is not necessarily cure, and that utilisation of services varies not only with sociodemographic factors, independent of the severity of symptoms and disability, but also with characteristics of the service, including factors relating to the doctors themselves (see for example Goldberg & Huxley, 1980). This variation greatly compromises the use of services as a proxy for outcome measures of health and leaves us no nearer our goal of measuring the impact of health care on the health of the population.

As we are in an era of relatively easy access to enormous quantities of data, it is vital to give critical thought to how useful the available data are for our purposes of measuring health and monitoring the activities of the health care system (Dean, 1988). It is better first to set priorities, identifying the major health objectives, then to choose the appropriate strategies, and only then to pick indicators to measure the resources that have been applied, how far the strategy has been put into action, and how far it is achieving its objective, than it is simply to use the data that are currently available and let them shape the questions we can answer. It is crucial to understand the meaning of a measure before interpreting it (Bergner *et al*, 1987). For example, one paper commented "The Goldberg score (derived from the GHQ28) may be considered as an indicator of a premorbid personality disposition towards depression" (Bucher & Gulzwiller, 1988). This is an entirely erroneous conclusion. The GHQ measures the presence of likely depression and anxiety, not personality traits. Indeed, it is specifically designed not to be contaminated by aspects of personality (Goldberg, 1978). Thus, in general, to answer a research question, it is far better to have understood the relationships between the theoretical concepts as far as possible and to use reasonably good indicators, than to use perfect indicators without knowing enough about their relationships (Mootz, 1988). Research must determine the way health is measured, not the availability of complex health indices. Otherwise, exclusive concentration on measurement will limit the conceptual development of the topic, and may inhibit thinking and planning.

Present outcome indicators in health care

The present health indicators that have been used as outcome measures include those of mortality, morbidity (as measured by disability days, bed days or restricted activity days, hospital admission figures), subjective health indicators, direct measures of health and social functioning, and measures of unmet need.

Mortality

This, the most frequently used measure, does not indicate health at all, but lack of survival (Dean, 1988). As a general principle, mortality is an important indicator of social conditions such as road safety and violence, as well as of the treatment of life-threatening diseases, but mortality data do not adequately reflect changes in health status within populations (Patrick & Erickson, 1987). Decreased mortality may not signal a healthier population; on the contrary, it may reflect the longer life of chronically ill and seriously disabled people (Wilson, 1981). In mental illness, there is of course an avoidable mortality, that caused by suicide. The official suicide rate, although known to be an underestimate, is a reasonable indicator of all deaths from suicide (Sainsbury, 1983), but it is not a good indicator at all of rates of mental illness. However, overall mortality rates, not just those of suicide, may be a helpful indicator of the adequacy of health care for mentally ill people. Fox & Goldblatt (1982) have demonstrated the grossly elevated standard mortality rates (SMRs) of people in the years following their discharge from mental hospital, largely due to cardiovascular and respiratory disease, presumably reflecting poor social and medical care.

Morbidity

Disability days, bed days, and restricted activity are indicators which may reflect many conditions other than health, such as provision for paid sick leave, the number and age of children in a family, and the division of responsibilities within families, as well as psychosocial factors such as the tendency to assume a sick role or to express problems through somatic symptoms. Disability days are more properly regarded as a measure of social disability (Wilson, 1981). However, they are useful, for example, as a measure of self-care during illness. (It is worth remembering that in a prospective investigation of 6928 adults, a pattern of greater mortality was found among persons with no disability days in comparison with those who reported one to three sick days during a year (Berkman, 1975); the greater mortality of people with no sick days was independent of age, sex, objective and subjective health status, and health habits.)

Bed usage and hospital admission/discharge figures are not in themselves outcome indicators because they are affected not only by the availability of beds, but also by:

(a) inflow factors, such as the availability of alternative services (e.g. day hospitals or community psychiatric nurses could reduce the need for admission)
(b) factors influencing length of treatment, such as the admission of heavily dependent patients when alternative arrangements in the community take a long time to set up, or the special interests of the team (e.g. a brief admission policy, the admission of forensic cases under a court order, in-patient psychotherapy)
(c) outflow factors, which influence the transfer of patients back to the community or to other appropriate units.

If these factors are explored and understood, then admission figures can become helpful to us in monitoring interventions to rationalise inflow, length of stay, and outflow. However, psychiatric in-patients represent only 1–2% of the total of psychiatric morbidity. In mental illness, as with other medical conditions of varying severity such as asthma or hypertension, not all cases reach hospital or even come to medical attention. It is therefore important for us to develop health indicators that apply not only to hospital in-patients, but also to the rest of the iceberg of morbidity in the community. Similarly, we need input and activity indicators not only for hospital services, but also for the primary-care services.

Subjective health indicators

These are measures that focus on experiential aspects of illness, distress and discomfort, instead of the more objective evaluation of health status by a professional. They are important to consider because of the research evidence that self-perceived health correlates with mortality and rate of recovery even after controlling for objective measures of health. Furthermore, subjective measures of health correlate more closely with use of health services than do the medical conditions themselves (Hunt, 1988). There is often a disparity between judgements of professionals and lay people on whether a particular treatment has been successful, and it is crucial to take the lay view as well as the medical view.

Direct measure of health and social functioning

These have by far the strongest conceptual basis as relevant indicators of health outcome. Many measures have separate physical, psychological, and social axes, while there are also combined measures, including measures of quality of life. It is not appropriate to describe these measures in detail here, but good reviews are available (e.g. McDowell & Newell, 1987; Thompson, 1989). Some measures assess positive health as opposed to ill health, presumably pursuing the World Health Organization's (1948) definition of health as "a state of complete physical, mental and social well being and not merely the absence of disease or infirmity". While the fact is that the National Health Service's overwhelming present task is to wrestle with the cure and prevention of illness and handicap, and with the delivery of care to the sick, and this should be its first objective, the pursuit of 'positive' health is not just a fanciful notion.

The importance of positive health is that it is typically used to describe some aspect of lifestyle or mental outlook that is associated with better future health. For example, a person with refined skills for coping with stress may not differ from others without such skills at the present time, but this person may have a higher probability of better mental and physical functioning in the future. However, it would be entirely premature to move from indicators of 'negative health' to those of 'positive health' (Bice, 1979).

Unmet need

'Unmet needs' have been defined as the differences between those services judged necessary to deal appropriately with defined health problems and those services actually being received. Changes in unmet need are measures of the outcome of health services, but they are not in themselves measures of health status (Carr & Wolfe, 1979). They do not measure the level of health of an individual or a population but rather they measure the capacity of the health and social care system to care for the sick. There is a strong tradition of measuring unmet need in the US, reviewed by Carr & Wolfe (1979), which has been developed by Brewin *et al* (1987, 1988) for mental illness. Indicators for unmet needs and health care outcome, although not the same, are related. Indicators for unmet needs relate to the services and resources necessary to maintain and improve health status. Carr & Wolfe (1979) suggested that the provision of appropriate health services according to need is an intermediate outcome of a health system whose ultimate goal may be to improve the health status of a population.

Global indicators

Some investigators have attempted to define a single 'gross national product' of health status, by combing data on mortality, morbidity, disability, and even health expenditure in various weighted ways into a health status index. Unfortunately such an approach tends to obscure rather than inform, because information about health must serve different purposes. In order to answer specific questions, the index has to be disaggregated into its component parts. It has been claimed that the loss of detail accompanying aggregation is offset by the increased generality of application. However, Bice (1979) argues that the history of measurement supports the view that progress is marked by increasing ability to discriminate between quantities and qualities of phenomena, and not by devising qualitative summaries that obscure differences.

Social indicators

The social dimension of health and the health care system must not be ignored, and Gulbinat (1983) pointed out that, in addition to direct measures of mental health, we need indicators of psychosocial development from childhood to old age, and also of social pathology (including family disruption and lack of social support). Some indicators of social pathology are readily available, such as the crime statistics and rates of divorce, illegitimacy, homelessness and unemployment, although there are the usual problems of interpretation, under-reporting, and changing cultural lifestyles, which negate the value of some official statistics (for example many couples may successfully cohabit for a number of years without entering the marriage statistics). Indicators of psychosocial development pose a much more difficult problem, but they should at least take into account age-specific and stage-specific problems, which have their own measurement requirements (Siegmann, 1979).

Demographic indicators

Finally, since health policy does not only aim to improve the average level of health but also to reduce inequalities in health among the various subgroups of a population, we not only need indicators of the level of health of the general population, but also an assessment of the differentials among population subgroups. This means that we also need a coherent strategy for applying health indicators across the nation, so that there is agreement on the subgroup definitions, which may be socioeconomic, ethnic, geographical, etc.

The need for research

While indicators can tell us about the quantity of mortality or morbidity, and can measure change over time, it is important to be clear that they cannot in themselves attribute change to a specific cause – it would be necessary to carry out carefully designed research projects which control for the other extraneous variables. Therefore it is vital to develop and test health indicators in the context of appropriate research projects to monitor health and health-promotion programmes (Culyer, 1983; Hansluwka, 1985; Lohr & Ware, 1987). Furthermore, there is a need for a closer dialogue between researchers and policy makers, leading to the notion of integrated health policy and research, in which responsibility is shared equally between the researcher and the end-user (Noack & McQueen, 1988).

A preliminary consideration of outcome indicators

This paper presents a brief consideration of indicators of health care input, process and outcome for schizophrenia, affective psychosis, neurosis, dementia, child psychiatry, forensic psychiatry, mental handicap, alcohol and drugs; it is not intended to be definitive or exhaustive but rather to form a basis for development by clinicians, researchers and planners for their own requirements.

Schizophrenia

Districts confront a life-time risk of schizophrenia of approximately 1% in the adult population, and while a third of cases do well, the remainder have repeated relapses and readmissions, and at least half of these undergo severe deterioration.

Before setting objectives, it is helpful to ask some preliminary basic questions.

Can the incidence of schizophrenia be reduced?

The aetiology of schizophrenia may be at least half genetic (Rosenthal, 1971), probably due to a polygenic rather than a single-gene mechanism (Cutting, 1985). Rainer (1982) quotes the life-time risk of developing the disorder as 10–15% for a child with one schizophrenic parent, and 35–40% for a child with two schizophrenic parents. Therefore adequate access to simple genetic counselling (by ensuring the education of primary-care and secondary-care teams to provide this on request) and the availability of contraceptive services may help a little in the long term (Reveley, 1985).

Can relapse rates and readmission rates be reduced?

Research has demonstrated that the delivery of family interventions (which reduce those family stresses and conflicts that precipitate relapse and readmission) and direct education of sufferers about the management of their own illness, can dramatically reduce relapse rates and readmission rates (Falloon *et al*, 1982, 1985; Leff *et al*, 1985; Birchwood *et al*, 1989).

Can total disability be reduced?

Research has demonstrated that the provision of adequate rehabilitative services can have a major impact on total disability (Wing, 1982), and that the accretion of severely dependent patients is much lower in the presence of such services (Hyde *et al*, 1987).

Is there an avoidable mortality?

Ten per cent of schizophrenics commit suicide (Hawton, 1987) and their SMRs are grossly elevated from those in general medical illness, including cardiovascular and respiratory diseases (Fox & Goldblatt, 1982), presumably contributed to by smoking, poor nutrition, poor environmental conditions, and lack of adequate medical and social care.

Objectives

These would therefore include:

(a) to reduce the incidence of schizophrenia
(b) to reduce relapse rates and readmission rates
(c) to reduce the total disability and deterioration in social functioning
(d) to prevent patients from 'falling through the care net'
(e) to reduce the suicide rate
(f) to reduce the premature mortality from all causes, especially cardiovascular and respiratory disease
(g) to reduce unemployment
(h) to provide satisfactory housing
(i) to continue research into the causes, consequences and care of schizophrenia.

Input indicators

These would be:

(a) genetic counselling services within the framework of both primary and secondary care
(b) social support for vulnerable people and families

(c) family interventions
(d) direct education of sufferers
(e) adequate rehabilitation services, including housing where necessary, and case-management systems
(f) education of primary-care and secondary-care teams about suicide risk
(g) adequate primary physical health monitoring
(h) adequate employment opportunities
(i) care registers to facilitate action and communication by secondary-care and primary-care teams
(j) active research on the causes, consequences and care of schizophrenia.

Process indicators

Indicators of activity on all the above factors would be required.

Outcome indicators

These would include:

(a) prevalence figures from the local psychiatric services register, together with grading of levels of disability, and supplemented by local-community research surveys
(b) hospital first-admission and readmission rates
(c) employment rates in local schizophrenic patients
(d) prevalence of suicide in local patients
(e) SMRs of patients with schizophrenia
(f) prevalence of homeless in local schizophrenic patients.

Affective psychosis

The life-time risk of affective psychosis is approximately 1% of the adult population; the illness is severe and recurrent, but it does not generally lead to deterioration between episodes. It can be treated and relapses can be minimised by the use of prophylactic drug therapy.

Again, before setting our objectives, it is helpful to ask some preliminary questions.

Can the incidence of affective psychosis be reduced?

The aetiology of affective psychosis is at least partially genetic (Reich *et al*, 1982). Morbid risk in relatives varies with unipolar and bipolar illness, age of onset, and responsiveness to treatment.

Can relapse rates and readmission rates be reduced?

These can be reduced by prophylactic drug therapy, and by adequate early-warning communication between family, primary-care and secondary-care teams and by the provision of adequate social support (Bennett, 1982).

Can total disability be reduced?

This disability is generally far less than for schizophrenia, as deterioration of personality does not occur in the same way, and multiple handicaps are less likely to accumulate. Adequate occupational opportunities are crucial after recovery (Bennett, 1982).

Is there an avoidable mortality?

Fifteen per cent commit suicide (Miles, 1977). Continuing education of primary-care and secondary-care teams in how to assess suicidal risk is vital if the rates are to be reduced.

Objectives

These would therefore include:

(a) to reduce the incidence of affective psychosis
(b) to reduce relapse rates and readmission rates
(c) to reduce the suicide rate
(d) to reduce the premature mortality from all causes
(e) to maximise employment
(f) to eradicate homelessness
(g) to continue research into the causes, consequences and management of affective psychosis.

Input indicators

These would be:

(a) genetic counselling services within the framework of primary and secondary care
(b) continuing education of primary-care team about risk and advantages of prophylactic drug therapy
(c) continuing education of primary-care and secondary-care teams about assessment of suicide
(d) adequate primary and physical health monitoring
(e) adequate employment opportunities
(f) care registers to facilitate action and communication by care teams
(g) active research into the causes, consequences and management of affective psychosis.

Process indicators

These would be indicators of activity on all the above factors.

Outcome indicators

These would include:

(a) prevalence figures from the local psychiatric services register, supplemented by local community research services
(b) hospital first-admission and readmission rates
(c) employment rates
(d) prevalence of suicide in local patients.

Neurosis

Districts confront depression and anxiety as very common disorders, occurring in about 10–25% of the adult population (depending on the threshold adopted for 'caseness'). Half recover in a few months, the remainder pursue a variable chronic course which may last two years or more. Although these conditions are not psychotic, and although some are minor, others are severe and handicapping, with damaging consequences for the sufferer and the wider community. Most should be readily treatable in primary care. Depression is used as an example.

Can the incidence of depression be reduced?

The causes of non-psychotic depression are environmental rather than genetic (Torgensen, 1983; Jenkins, 1985). Some depression is secondary to physical disease, pain and disability, and is helped by the prevention and treatment of physical disease, pain and disability (particularly loss of mobility and sensory function) (e.g. Cooper, 1976). Some cases of depression are precipitated by acute life events and can be prevented by crisis intervention, and other systems of support (Newton, 1988). Some is precipitated by lack of social support and can be prevented by setting up systems for at-risk groups (e.g. isolated mothers of pre-school children). Organisations such as CRUSE and NEWPIN, as well as adequate child and family psychiatric services, may all help.

Can recovery rate be improved?

The prognosis can be improved by improving the detection and treatment of depression in general practice. Research has shown that depression detected by the general practitioner (GP) has a better prognosis than 'hidden' morbidity (Johnstone & Goldberg, 1976). At present GPs only identify about half of the total morbidity (Goldberg & Huxley, 1980).

Is there an avoidable mortality?

A small proportion of depressed people commit suicide, and the risk of death from all causes including accidents is twice the norm in severe neurotic depression (Sims & Prior, 1978).

Objectives

These would therefore include:

(a) to reduce the incidence of depression
(b) to increase detection rates by primary-care teams
(c) to reduce chronicity of depression
(d) to reduce the premature mortality from all causes
(e) to continue research into the causes, consequences and care of depression, particularly in primary care.

Input indicators

These would be:

(a) adequate screening and treatment facilities in primary care for physical disease and disability, loss of morbidity and sensory function
(b) system for primary-care teams to identify those in their practice populations who are particularly at risk, including the elderly, young, isolated mothers of pre-school children, the disabled, and informal carers of severely physically ill or mentally ill patients, and to screen regularly, either opportunistically, or on home visits where appropriate
(c) resource indexes in primary care, listing sources of support for their practice population
(d) counsellors, psychologists and social workers in primary health care
(e) 'stress' programmes in local workplaces, with good collaboration between industry and health care teams
(f) adequate training in detection (and treatment where appropriate) of depression for primary-care teams, social services, and occupational health services
(g) active research studies on the causes, consequences and care of depression, particularly in primary care.

Process indicators

These would be indicators of activity on all the above factors.

Outcome indicators

These would be:

(a) community surveys
(b) general practice identification index
(c) prevalence of suicide and parasuicide.

Dementia

Dementia involves an irreversible decline in cognitive faculties, ending in death. It occurs in 5–10% of all adults over the age of 65, and increases with age, occurring in 20% of those aged over 80.

Can incidence be reduced?

The incidence of arteriosclerotic dementia can be reduced by factors reducing arteriosclerosis. The incidence of senile dementia cannot at present be reduced.

Can functioning be improved?

Functioning of demented people can be improved significantly by various general measures: appropriate support for living in the community, to prevent admission to institutions with its attendant risks of decompensation of dementia, depression, decreased mobility, and death; and adequate training and support for carers in the community and in institutions, particularly in the recognition of depression and toxic confusional states.

Is there an avoidable mortality?

Life expectancy is five years for senile dementia, and two years for arteriosclerotic dementia, and at present little can be done to improve mortality rates.

Objectives

These would include:

(a) to maximise functioning
(b) to reduce the incidence of depression and toxic confusional states
(c) to ensure adequate training and support to all carers, both formal and informal
(d) to continue research into the causes, consequences and care of senile dementia.

Input indicators

These would include:

(a) adequate support services (e.g. home-helps, laundry services, incontinence services, meals, medical checks, attendance at day centres, lunch clubs, adequate chiropody, physiotherapy, and mobility, aids for deafness and blindness)
(b) night nursing and respite care, to support informal carers in their task
(c) adequate training and support for informal and formal carers, particularly in recognising treatable conditions such as depression and toxic confusional states which will otherwise greatly reduce functioning
(d) availability of a range of institutional care when necessary (e.g. Part-III accommodation, nursing homes, day care)
(e) active research in the causes, consequences and care of senile dementia.

Process indicators

These would include indicators of activity on all of the above.

Outcome indicators

These would include:

(a) community and institutional surveys of prevalence and severity of dementia, depression and toxic confusional states
(b) admissions to institutional care and day care.

Child psychiatry

The two major disorders of childhood are emotional disorders and conduct disorders (occurring in one in ten children and one in five adolescents) (Rutter, 1989). Psychosis in childhood is very rare (less than 1 per 1000).

External influences are as important as the child's temperament and character. Clearly, individuals differ in their resilience to environmental stresses. Different undesirable experiences may have different risk potentials for cognitive, socio-emotional and behavioural development (Kolvin *et al*, 1990). In the last two decades the developmental process has been seen as more fluid than before, and dependent on the interaction of genetic and environmental processes at all stages, from the intrauterine environment to the psychosocial environment of the school. There has been a considerable growth in research into both the genetic and environmental transmission of child psychiatric disorder (Earls & Jung, 1987).

Can incidence be reduced?

Sociocultural factors play an important role in the incidence of emotional and conduct disorders, and it is likely the greater our understanding of the way such factors influence emotion and behaviour, the easier it will be to reduce their incidence.

Mentally handicapped children have a higher rate of psychiatric disorder, possibly three to four times higher than that found in a randomly selected control group (Rutter *et al*, 1970*a*,*b*). Factors lowering the incidence of mental handicap will lower the incidence of psychiatric disorder overall.

The child psychiatric disorders that follow head injury can be reduced in incidence by improved prevention of head injury, for example by wearing an adequate helmet when cycling, better car seat-belt legislation, etc. We know some of the specific risk factors with regard to psychiatric disorder in childhood and adolescence, such as organic brain dysfunction, lead toxicity, and family discord. We know that the school and the area of residence both have influences on the onset of psychiatic disorder.

Other evidence suggests that environmental deprivation can be prevented by a network of social and emotional supports, especially in the pre-school years, ensuring good maternal health, an absence of financial and employment problems, encouraging a small family size, and helping women to wait until they are emotionally mature before becoming mothers.

Can relapse and readmission rates be reduced?

Very few children in fact require hospital admission. Rates are subject to the same factors as in general adult psychiatry. However, disturbed children and adolescents may be admitted to educational or social services facilities as well as hospitals.

Can total disability be reduced?

There is evidence that total disability can be reduced, but not to what extent.

Is there an avoidable mortality?

The two psychiatric conditions most associated with death in this age group are suicide and overt anorexia nervosa, although both are rare. Death from child abuse is related to parental psychopathology, but abused children may be referred to a child and adolescent psychiatry service (those deaths are usually prevented by vigorous interventions by health, social services and the court).

Major depression and suicide constitute an increasing public health problem among the young. Risk factors for suicide are: male sex, depression, intoxication, previous suicide attempts, and ready access to the means. Early detection and amelioration of depression, appropriate and early intervention for parasuicide, and controlling the commonly used methods of suicide (e.g. drugs and guns) are likely to reduce the suicide rate.

Objectives

These would include:

(a) to reduce the incidence of psychiatric disturbance
(b) to reduce rates of relapse, where applicable
(c) to reduce the rates of hospital readmission
(d) to reduce the parasuicide rate
(e) to reduce the suicide rate

(f) to reduce educational failure due to psychiatric disturbance
(g) to improve the quality of life for those children caught in psychosocially disadvantaged families
(h) to reduce the impact of social deprivation and emotional deprivation
(i) to improve the detection of those likely to develop chronic illness
(j) to continue research in this specialty.

Input indicators

These would include:

(a) improved training of medical undergraduates and GP postgraduate training to enhance the ability of GPs to detect psychiatric disturbance in children and adolescents and to know when to refer those to specialist services
(b) provision by health visitors of a service to families with pre-school-aged children, as by nursery nurses, play-group leaders
(c) provision by teachers, school welfare officers and educational psychologists of a service to school children (and some identified pre-school children), as by the school health service employees
(d) provision by social workers of counselling services to families, and their statutory duties to provide care and protection for some children at risk
(e) specialist services offered by child and adolescent psychiatrists, child psychotherapists, clinical and educational psychologists, and other therapists, e.g. speech therapists, physiotherapists, drama therapists
(f) specialist health facilities, including out-patient, day-patient and in-patient services, on a district, regional or supraregional basis, according to need as well as financial and manpower resources (of special note are the two youth treatment centres – the adolescent forensic service provided by the Gardner Unit at Prestwich Hospital, and the Young Persons Unit in Ashworth Hospital)
(g) additional specialist educational services, such as special day and residential schools
(h) additional specialist social services, such as community homes (with or without education provided on the premises), assessment centres, the Aycliffe Institute for Adolescent Disorders
(i) independent organisations providing counselling and psychotherapy services, in-patient and out-patient services and residential homes
(j) extensive research conducted nationally and internationally.

Process indicators

These include indicators on activity on all the above factors.

Outcome indicators

Outcome, in this field, is not always measurable clinically; for example a request to write a report for the court will have a legal outcome. Often the outcome is a *social* one and not a health one. Change can be seen as 'dynamic' change or 'symptom' change. Some indicators are:

(a) measures of function (a dynamic change, e.g. learning better)
(b) symptoms reduced in severity or frequency
(c) symptoms removed
(d) prevalence rate reduced
(e) rate of attempted suicide reduced
(f) rate of completed suicide reduced
(g) chronic disturbance detected at an early stage
(h) readmission rate for serious disorders reduced
(i) rate of reception into care reduced (a social outcome)
(j) delinquency prevented or the rate of police involvement/court appearances reduced.

Forensic psychiatry

District health authorities have a responsibility for the care of mentally disordered offenders who originate from their district. The prevalence of mentally disordered offenders in the general population is unknown, although the prevalence in prison populations has been variously described as between 4% and 20%. Of the resident population in all mental illness and mental handicap hospitals on 31 December 1986, 2.5% were detained under the criminal provisions of the Mental Health Act 1983 (unpublished Home Office calculation). A relatively small group of psychiatric patients have many problems: unemployment, homelessness, often intractable and severe mental disorder which has left social handicaps, behavioural problems, and potential for reoffending. As with all ex-psychiatric patients, the SMRs are elevated. This group often requires management by a combination of psychiatric teams, probation services, prison medical service, social work, general medical and general practitioner teams, as well as appropriate liaison with police, courts, Crown Prosecution Service and solicitors acting on behalf of their clients.

Can the numbers of mentally disordered offenders be reduced?

Mental illness (above) and mental handicap (below) are discussed in other sections, so this section concentrates on personality disorder (the commonest psychiatric condition associated with persistent offending), where it is possible that incidence could be reduced. Illegal delinquent acts are common (80% of all boys). Only a minority become persistent adult offenders. A number of environmental factors significantly increase the chance of becoming persistently delinquent: bad parenting, criminal fathers, alcoholic parents, low IQ, low family income, and large family (West & Farrington, 1977), and school (Rutter & Giller, 1983). Early identification of vulnerable people and families with appropriate interventions by caring agencies (training for parenthood, medical help for alcohol problems, social services and probation services support, school awareness, etc.) may help to reduce the incidence. Rutter & Giller (1983) showed that a number of factors may protect a person against delinquency: a good peer group (e.g. at school), successful employment, a good marriage, an improvement in life circumstances, one good relationship, good life experience outside the home (e.g. at school).

Can relapse rates and readmission rates be reduced?

For personality disorders, counselling and general support (probation and out-patient services) seem to help, although this is stated on the basis of clinical experience rather than research. It certainly seems to assist in preventing reoffending, in some people. Patients may well develop insights into their difficulties, which will help them successfully manage stressful events in their lives.

For mentally disordered offenders, careful follow-up after discharge from a regional secure unit has been shown to result in less time spent in hospital and less reoffending during the two years after admission compared with the two years before (unpublished DHSS study).

Can total disability be reduced?

There is a tendency for maturation to take place with time. This is probably helped in some cases by general support and counselling, which lead to greater ability on the part of the patient to cope. Treatment in special therapeutic communities (e.g. Grendon Prison) is thought to help, especially with younger, more intelligent psychopaths – but again, there is not a controlled trial to establish this. Research suggests that on return to the original environment, the benefits of the more structured setting are lost (Gunn *et al*, 1978).

Is there an avoidable mortality?

About 5% of people with psychopathic disorder commit suicide. Those with personality disorder are often said to be more reckless and to have a higher death rate from accidents.

Objectives

These would include all the objectives given under 'Schizophrenia' and 'Mental handicap'. Additional objectives would include:

(a) reduce the incidence of mentally disordered offenders
(b) reduce the incidence of personality disorder
(c) reduce reoffending rates, as well as relapse and readmission rates
(d) reduce suicide rates
(e) reduce premature mortality
(f) prevent entry and re-entry into criminal justice system
(g) reduce the social handicaps
(h) reduce unemployment
(i) reduce homelessness.

Input indicators

These would include, as well as those for schizophrenia and mental handicap:

(a) systems to identify vulnerable people and families, and deliver social support, health services, probation services, school support, and pre-parenthood training
(b) systems to provide psychiatric services for assessment, and advice to courts, police stations, Crown Prosecution Service, probation services, and to provide early diversion from the criminal justice system
(c) systems to provide psychiatric service to prisons, to aid transfer of mentally disordered prisoners to hospital
(d) systems to ensure ongoing support and care from psychiatric and other services on release from prison
(e) systems to ensure rehabilitation, and to facilitate multi-agency management (e.g. assessment panels)
(f) access to housing
(g) adequate employment opportunities.

Process indicators

These would be indicators of activity on all above factors.

Outcome indicators

These would include:

(a) numbers of patients detained under Section IV of the Mental Health Act 1983, and their admission and readmission rates
(b) prevalence of treatable mentally disordered offenders in prison population
(c) numbers of patients diverted from criminal justice system (not collected at present)
(d) suicide rate in prison (overall rate in 1987 was 39 per 50 265 prisoners)
(e) SMRs.

Mental handicap

The prevalence of mental handicap varies according to the degree of handicap and to age, but an overall prevalence of severe mental handicap is 3–4 per 1000. Mild mental handicap is found in 20–30 per 1000. The incidence of severe mental handicap is 5.4 per 1000 live births. It is thought that 1.5 per 1000 people with mental handicap are at risk of developing a mental illness (Corbett, 1979). Day (1983) suggested 0.25 beds per 1000 people were required for a comprehensive psychiatric service for mental handicap.

Most mentally handicapped people have the same health care needs as the rest of the population, but some conditions are prone to the development of specific illnesses, such as thyroid dysfunction in people with Down's syndrome. The mentally handicapped seem more prone to developing psychiatric disturbance (with an incidence as high as 50–60%). As longevity is increasing in the mentally handicapped, special problems are being found in the elderly, such as an increased risk of fractures. Special provision must be made, such as respite care facilities, special education, adult training centres, special education (resource) centres, advocates and brokers, and specialist health services to meet special needs, on top of the primary-care services.

Can incidence be reduced?

A third of cases have no known cause. The remaining two-thirds could be reduced by the following measures:

(a) genetic counselling
(b) screening for inherited disorders (including infections)

(c) vaccination, where available, for congenital infections
(d) health education directed at young women, concerning nutrition, smoking, and alcohol consumption during pregnancy
(e) control of exposure of parents/foetus to toxins (e.g. proposed link between men exposed to hydrocarbons and offspring with Prader–Willi syndrome)
(f) avoid hazards of birth process and improved neonatal care, especially of low- and very-low-birthweight babies
(g) measures to reduce brain-damaging accidents in childhood.

Reducing the incidence of mental illness in the mentally handicapped is the same as for mental illness generally.

Can relapse and readmission rates be reduced?

The early detection of mental illness in the severely mentally handicapped requires special skills because of the communication problems. Prompt management reduces the likelihood of secondary handicaps and the need for admission.

Can total disability be reduced?

Total disability can be reduced by the following measures:

(a) detection of metabolic disorders in infancy, with prompt and adequate treatment of detected cases, to prevent the onset of mental handicap or reduce its impact
(b) detection and appropriate treatment of serious physical illness at an early stage
(c) early detection of sensory defects
(d) early and continuing appropriate remedial education, not necessarily ending at 16
(e) fostering self-help and independence and preventing institutionalisation
(f) providing adequate and appropriate day-care facilities
(g) support for carers
(h) regular monitoring of nutritional status, including minerals and vitamins, and care in the prescribing of psychotropic drugs in the elderly mentally handicapped, to minimise fractures.

Is there an avoidable mortality?

The fact that longevity is increasing in the mentally handicapped is proof that there is avoidable mortality. One continuing problem in the elderly mentally handicapped is respiratory-tract infections and, although deaths from status epilepticus have decreased, deaths due to carcinoma (particularly gastrointestinal), myocardial infarction, and cerebrovascular accident have increased (Jancar, 1988). Improved mortality is due to new antibiotics and anticonvulsants, better diet and care and better environment (Jancar, 1988).

Objectives

These would include:

(a) to ensure all people with a mental handicap receive the same health care as the rest of the population
(b) to ensure that additional special needs receive appropriate services
(c) to ensure that the incidence of mental handicap is reduced
(d) to ensure that prompt detection and appropriate management of serious physical or mental illness occurs to avoid hospital admission or readmission
(e) to ensure that additional disability is prevented
(f) to continue, through research, to prevent avoidable early death
(g) to improve self-esteem and reduce psychiatric morbidity by encouraging improved education attainments and social skills training
(h) to care for the carers, to enable them to continue to care.

Input indicators

These would include:

(a) to foster the widespread establishment of mental handicap registers
(b) to establish enrolment of each person with a mental handicap on the list of a GP
(c) to ensure each DHA has a written policy on the health care provision for the mentally handicapped, including specialist services to meet special needs such as medical and nursing psychiatric care, speech therapy (now in drastically short supply), physiotherapy, occupational therapy – including medical audit
(d) regular assessment of sight and hearing
(e) genetic counselling and continued research in genetics
(f) screening for inherited disorders
(g) vaccination availability and improve uptake
(h) health education, including accident prevention, effects of alcohol and nicotine in pregnancy, etc.
(i) control of environmental hazards
(j) high standards of obstetric and neonatal care
(k) continued research into the special needs of the elderly mentally handicapped
(l) regular monitoring of minerals and vitamins in the elderly mentally handicapped
(m) to ensure appropriate day-care facilities are available, and to improve on work opportunities
(n) to continue the programme of community care to provide as normal a living environment as possible
(o) to improve educational services to help a person reach optimum ability
(p) availability of respite care services to lighten the burden on carers.

Process indicators

Indicators of activity on all the input indicators, including research on how to identify consumer satisfaction, would be required.

Outcome indicators

These would include:

(a) prevalence figures for males and females, for mild/moderate and severe mental handicap, for specific syndromes, and the total

(b) statistics on brain-damaged children left mentally handicapped
(c) statistics on the age at death for men and women as well as the cause of death
(d) hospital admission and readmission rates
(e) statistics on additional handicaps such as sensory defects, behaviour disturbance, motor disability
(f) levels of obtained employment, uptake of day services
(g) improved uptake of respite care services
(h) estimates of consumer satisfaction (possibly by questionnaires)
(i) a record of the registers available nationally.

Alcohol misuse

The incidence depends on the definition of what constitutes misuse. For example, 6% men and 1% women are very heavy drinkers, while 25% men and 9% women are drinking more than agreed 'sensible' levels. The consequences of alcohol misuse may be physical, psychological and social, and arise both from chronic alcohol misuse (e.g. cirrhosis) and from acute intoxication (e.g. road accidents).

Can incidence be reduced?

The incidence of alcohol misuse can be reduced by a wide range of factors which influence either availability or demand.

Can relapse rate and hospital readmission rates be reduced?

Relapse rates and readmission rates can be reduced. The best chance of success is when the problem drinking is at an early stage. Minimal intervention (simple advice, brief follow-up, and feed-back on progress) has a high rate of success at this stage, particularly if the family can be involved. Improvement is much lower for severe chronic drinking problems.

Can total disability be reduced?

While some of the pathological changes caused by excessive alcohol consumption are irreversible, a remarkable degree of physiological recovery can occur with abstinence and attention to nutrition and vitamins. Problem drinkers may have special accommodation needs, require medical treatment to prevent further physical disability or continued support to stop or reduce drinking.

Is there an avoidable mortality?

Alcohol-related accidents and illness cause around 7000 deaths each year and 15% of alcoholics commit suicide.

Objectives

These would include:

(a) to reduce the prevalence of alcohol misuse
(b) to reduce relapse and readmission rates with alcohol-related diagnoses
(c) to reduce suicide rates
(d) to reduce premature mortality, especially from chronic liver disease
(e) to eradicate or reduce homelessness among people who misuse alcohol.

Input indicators

These would be:

(a) education and support for primary-care teams to encourage screening, detection and better management of problem drinkers
(b) continuing education of primary- and secondary-care teams about assessment of suicide
(c) provision of a range of services including advice centres, day centres, detoxification facilities (home and residential), hostel accommodation, hospital-based services, and community-based services (e.g. community alcohol team)
(d) case register.

Outcome indicators

These would be:

(a) hospital admissions, discharges and deaths with a diagnosis of alcohol-related mental illness or liver disease
(b) local alcohol consumption - level and pattern (obtainable by community survey)
(c) prevalence of suicide in local patients with alcohol misuse
(d) prevalence figures from local case register.

Drug misuse

The absolute number of drug misusers is unknown. It has been estimated that nationally there are 75 000–100 000 misusers of opiate drugs, and as many again who misuse a variety of other illicit drugs (Department of Health and Social Security, 1988, 1989). This estimate excludes cannabis users (possibly three million nationally).

Research has suggested that over ten years, 40% of illicit opiate users will become abstinent, one-third will achieve greater social stability, and the remainder will continue chaotic use or die. There is an annual mortality of 200–300. Illicit drug users have high rates of accident, and accidental overdose. Injecting drug use with shared injecting equipment is associated with 3% of reported AIDS cases nationally, and an increasing proportion of reported HIV-infected people (currently 10%) (Director of Public Health Laboratory Service, 1990). Social and relationship problems are common, and in some areas there is well researched association with unemployment and homelessness. Drug-related legal problems further disadvantage the user. Management of the problematic drug user must therefore contain not only psychiatric but medical and social elements of care.

Can incidence be reduced?

Law enforcement has a major role in reducing the availability of illicit drugs. Health education on the effects of drugs, and the appropriate use of medication, can improve information about drugs and their use or misuse, but there is no clear evidence in the international research literature that drug education necessarily has any long-term effect on illicit drug taking. There is, however, some recent evidence that information and education about the HIV-related risks of sharing needles may influence people (Stimson *et al*, 1988). Benzodiazepine dependence can be reduced by careful assessment and prescribing. The illicit misuse of other pharmaceutical drugs can be reduced by good security in the storage and careful prescribing of controlled drugs.

Can relapse rates and readmission rates be reduced?

Early intervention and harm-minimisation strategies can reduce physical morbidity and legal involvement. Habilitative strategies particularly for young adolescent users, including opportunities for education, training and employment, may reduce continued misuse and relapse (d'Orban, 1982). For chronic users, similar rehabilitative strategies, and the provision of adequate housing, can improve stability, and provide a basis for appropriate domiciliary care should HIV infection supervene.

Can total disability be reduced?

Provision of adequate rehabilitative services and long-term support can improve social and health functioning, even in the absence of abstinence.

Is there an avoidable mortality?

Drug users may lack access to primary health care, leading to unnecessary morbidity and mortality from usually non-fatal conditions. In chronic users immunosuppression related to drug use *per se* and to injecting has been demonstrated. Health education about the risks of shared injecting may reduce the risks of HIV infection and hepatitis B. Drug users following involuntary withdrawal (as in prison) are at higher risk of death from accidental overdose, due to loss of tolerance following release. Clear information and health education before release are necessary. Controlled substitution prescribing reduces the risk of mortality from use of illicit, contaminated drugs.

Objectives

These would include:

(a) to reduce the incidence of prescribed problematic drug use
(b) to reduce relapse rates and readmissions
(c) to reduce deterioration in social functioning
(d) to reduce the rate of accidental overdose and infections (including HIV)
(e) to reduce mortality
(f) to reduce overall health morbidity
(g) to reduce unemployment
(h) to reduce inadequate housing

Input indicators

These would be:

(a) accessible and available primary care services
(b) community-based specialist drug agencies providing a range of treatment options
(c) designated secondary-level hospital provision
(d) systems of social support to users and their families
(e) appropriate health education for users and their families
(f) adequate rehabilitation services, including hostels, half-way houses, and housing
(g) specific harm-minimisation strategies, needle-exchange projects, outreach work
(h) adequate training, education and employment opportunities
(i) training for all professionals likely to make contact with drug users.

Process indicators

These would be indicators of activity on all the above factors.

Outcome indicators

These would include:

(a) incidence of prevalence data from the regional drug database and the Home Office Addicts Index, supplemented by local-community research surveys
(b) hospital admission and readmission rates for all drug-related conditions
(c) attendances at designated day centres
(d) contacts with community psychiatric nurses
(e) employment rates of drug users
(f) prevalence of HIV and hepatitis B in injecting drug users, their sexual partners and children
(g) local drug-related offence rate
(h) coroner's records of accidental overdose.

Conclusion

At present, most data from the Department of Health are gathered on indicators of process. As financial resources are at such a premium, it would seem more useful to concentrate them on measuring inputs and outcomes first of all, and only to use process where necessary, to investigate shortfalls in achieving desired objectives.

The suggestions for health indicators here are not in any way intended to be exhaustive, but rather to serve as pointers towards the kind of measurement which clinicians, researchers, health authorities, family practitioner committees and DPHs can usefully engage in to fulfil their objectives of assessing quality of health care. There are opportunities for collaboration between secondary care services and DPHs (referred to earlier), and also between primary-care and secondary-care services. For example, GPs, as part of their new contract, are required to screen patients over 75 annually. Such a screen should properly include scales for dementia and depression, and therefore collaboration with the secondary-care services before and after data collection would be mutually beneficial.

Acknowledgements

I am greatly indebted to my colleagues Dr D. Black, Dr D. Brooksbank, Dr D. Jones, Dr H. Markowe, Dr P. Mason, Dr J. Reed and Dr J. Shanks and Mr R. Anderson at the Department of Health for helpful advice on earlier versions of this manuscript, and Miss Lynette Timms for secretarial assistance.

References

Bennett, D. H. (1982) Management and rehabilitation of affective psychoses. In *Handbook of Psychiatry, vol. 3, Psychoses of Uncertain Aetiology* (eds J. K. Wing & L. Wing). Cambridge: Cambridge University Press.

Bergner, M., Bobbit, R. A., Kressel, S., *et al* (1979) The sickness impact profile: conceptual formulation and methodology for the development of a health status measure. In *Sociomedical Health Indicators* (eds J. Elinson & A. E. Siegmann). New York: Baywood.

——, Kaplan, R. M. & Ware, J. E. (1987) Evaluating health measures. Commentary: measuring overall health: an evaluation of three important approaches. *Journal of Chronic Diseases*, **40** (suppl.), 235–265.

Berkman, P. (1975) Survival and a modicum of indulgence in the sick role. *Medical Care*, **13**, 85.

Bice, T. W. (1979) Comments on health indicators. Methodological perspectives. In *Sociomedical Health Indicators* eds J. Elinson & A. E. Siegmann). New York: Baywood.

Birchwood, M., Smith, J., Macmillan, S., *et al* (1989) Predicting relapse in schizophrenia: the development and implementation of an early signs monitoring system using patients and families as observers. *Psychological Medicine*, **19**, 649–656.

Brewin, C. R., Wing, J. K., Mangin, S P., *et al* (1987) Principles and practice of measuring needs in the longterm mentally ill; the MRC Needs for Care Assessment. *Psychological Medicine*, **17**, 971–981.

——, ——, ——, *et al* (1988) Needs for care – the longterm mentally ill; a report from the Camberwell High Contact Survey *Psychological Medicine*, **18**, 457–468.

Bucher, H. & Gutzwiller, F. (1988) Evaluating an indicator of preventive health behaviour derived from a national study. *Health Promotion*, **3**, 73–78.

Carr, W. & Wolfe, S. (1979) Unmet needs as social indicators. In *Sociomedical Health indicators* (eds J. Elinson & A. E. Siegmann). New York: Baywood.

Cook, T. D. & Campbell, D. T. (1976) The design and conduct of quasi experiments and true experiments in field settings. In *Handbook of Industrial and Organisational Psychology* (ed. M. D. Dunnette). Chicago: Rand McNally.

Cooper, A. F. (1976) Deafness and psychiatric illness. *British Journal of Psychiatry*, **129**, 216–226.

Corbett, J. A. (1979) Psychiatric morbidity and mental retardation. In *Psychiatric Illness and Mental Handicap* (eds F. E. James & R. P. Snaith). London: Gaskell.

Culyer, A. J. (1983) *Health Indicators An International Study for the European Science Foundation*. Oxford: Martin Robertson.

Cutting, J. (1985) *The Psychology of Schizophrenia*. Edinburgh: Churchill Livingstone.

Day, K. (1983) A hospital based psychiatric unit for mentally handicapped adults. *Mental Handicap*, **11**, 137–140.

Dean, K. (1988) Issues in the development of health promotion indicators. *Health Promotion*, **3**, 13–21.

Dell, S. & Gibbens, T. C N. (1971) Remands of women offenders for medical reports. *Medicine, Science and the Law*, **11**, 117–127.

Department of Health and Social Security (1988) *On the State of the Public Health for the Year 1987*. London: HMSO.

—— (1988) *Aids and Drug Misuse Part I. Report by the Advisory Council on the Misuse of Drugs*. London: HMSO

—— (1989) *Aids and Drug Misuse Part II. Report by the Advisory Council on the Misuse of Drugs*. London: HMSO.

Director of the Public Health Laboratory Service (1990) Acquired immune deficiency syndrome in England and Wales to end 1993; projections using data to end September 1989. *Communicable Disease Report, January 1990*. London: Public Health Laboratory Service.

D'Orban, P. (1982) Drug dependence. In *Handbook of Psychiatry, vol. 4 The Neuroses and Personality Disorders*. (eds G. F. M. Russell & L. A. Hersov). Cambridge: Cambridge University Press.

Earls, F. & Jung, K. G. (1987) Temperament and home environment characteristics as causal factors in the early development of childhood psychopathology. *Journal of the American Academy of Child and Adolescent Psychiatry*, **26**, 491–498.

Falloon, I. R. H , Boyd, J. L., McGill, C. W , *et al* (1982) Family management in the prevention of exacerbations of schizophrenia. *New England Journal of Medicine*, **306**, 1437–1440.

——, ——, ——, *et al* (1985) Family management in the prevention of morbidity of schizophrenia: clinical outcome of a two year longitudinal study. *Archives of General Psychiatry*, **42**, 887–896.

Fox, A. J. & Goldblatt, P. O. (1982) *Longitudinal Study – Sociodemographic Morality Differential*, LS No. 1, 1971–1975. London: HMSO.

Goldberg, D. (1978) *Manual of the General Health Questionnaire*. Windsor: NFER.

—— & Huxley, P. (1980) *Mental Illness in the Community: The Pathway to Psychiatric Care*. London: Tavistock.

Gulbinat, W. (1983) Mental health problem assessment and information support direction of WHO's work. *World Health Statistics Quarterly*, **36**, 224–233.

Gunn, J., Robertson, G., Dell, S., *et al* (1978) *Psychiatric Aspects of Imprisonment*. London· Academic Press.

Hansluwka, H. E. (1985) Measuring the health of populations, indicators and interpretations. *Social Science and Medicine*, **20**, 1207–1224.

Hawton, K. (1987) Assessment of suicide risk. *British Journal of Psychiatry*, **150**, 145–153.

Hunt, S. M. (1988) Subjective health indicators and health promotion. *Health Promotion*, **3**, 23–24.

Hyde, C., Bridges, K., Goldberg, D. *et al* (1987) Evaluation of a hostel ward. A controlled study using modified cost benefit analysis. *British Journal of Psychiatry*, **151**, 805–812.

Jancar, J. (1988) Consequences of longer life for the mentally handicapped. *Geriatric Medicine*, **18**, 81–87.

Jenkins, R. (1985) Sex differences in minor psychiatric morbidity. *Psychological Medicine* (monograph suppl. 7).

Johnstone, A & Goldberg, D. (1976) Psychiatric screening in general practice. *Lancet*, *i*, 605–608.

Kolvin, I., Miller, F. J. W., Scott, D. M., *et al* (1990) Continuities of deprivation? The Newcastle 1000 family study ESRC/DHSS *Studies in Deprivation and Disadvantage*, **15**, 379–393.

Leff, J., Kuipers, L., Berkowitz, R., *et al* (1985) A controlled trial of social intervention in the families of schizophrenia patients: two-year follow up. *British Journal of Psychiatry*, **146**, 594–600.

Lohr, K. N. & Ware, J. E., Jr (eds) (1987) Proceedings of the advances in health assessment conference. *Journal of Chronic Diseases*, **40** (suppl. 1)

McDowell, I. & Newell, C. (1987) *Measuring Health: A Guide to Rating Scales and Questionnaires*. Oxford: Oxford University Press.

Miles, C P. (1977) Conditions predisposing to suicide: a review. *Journal of Nervous and Mental Diseases*, **164**, 231–246.

Mootz, M. (1988) Health (promotion) indicators; realistic and unrealistic expectations. *Health Promotion*, **3**, 79–84.

Newton, J. (1988) Approaches to prevention: supporting troubled persons. In *Preventing Mental Illness*. London: Routledge & Kegan Paul.

Noack, H. & McQueen, D. (1988) Towards health promotion indicators. *Health Promotion*, **3**, 73–78.

Patrick, D. & Erickson, P. (1987) *Assessing Health Related Quality of Life in General Population Surveys: Issues and Recommendations*. Washington, DC: National Center for Health Statistics

Rainer, J. D. (1982) Genetics of schizophrenia In *Handbook of Psychiatry, vol 3 Psychoses of Uncertain Aetiology* (eds J. K. Wing & L Wing) Cambridge: Cambridge University Press.

Reich, T., Cloninger, C. R., Suarez, B., *et al* (1982) Genetics of the affective psychoses In *Handbook of Psychiatry, vol. 3. Psychoses of Uncertain Aetiology* (eds J. K. Wing & L. Wing). Cambridge: Cambridge University Press.

Reveley, A (1985) Genetic counselling for schizophrenia. *British Journal of Psychiatry*, **147**, 107–112.

Rosenthal, D. (1971) *Genetics of Psychopathology*. New York McGraw Hill.

Rutter, M (1989) Isle of Wight revisited: twenty-five years of child psychiatric epidemiology. *Journal of the American Academy of Child and Adolescent Psychiatry*, **28**, 633–653.

——, Graham, P. & Yule, W. (1970*a*) *A Neuropsychiatric Study in Childhood*. London: SIMP/Heinemann

——, Tizard, J. & Whitmore, K , (eds) (1970*b*) *Education, Health and Behaviour*. London: Longman.

Rutter, M. & Giller, H. (1983) *Juvenile Delinquency – Trends and Perspectives*. Harmondsworth: Penguin.

Sainsbury, P. (1983) Validity and reliability of trends in suicide statistics. *World Health Statistics Quarterly*, **36**, 339–348

Shaw, C. D. (1989) Audit in internal medicine. *British Journal of Hospital Medicine*, **42**, 19.

Siegmann, A. E. (1979) A classification of sociomedical health indicators: perspectives for health administrators and health planners. In *Sociomedical Health Indicators* (eds J. Elinson & A. E. Siegmann). New York: Baywood.

Sims, A & Prior, P. (1978) The pattern of mortality in severe neuroses. *British Journal of Psychiatry*, **133**, 299–305

Stimson, G. V., Alldritt, L., Dolan, K., *et al* (1988) *Injecting Equipment Exchange Schemes: Final Report* London. Goldsmith's College

Thompson, C. (ed.) (1989) *The Instruments of Psychiatric Research*. Chichester: Wiley.

Torgensen, S (1983) Genetics of neuroses: the effects of sampling variation upon the twin concordance ratio. *British Journal of Psychiatry*, **142**, 126–132.

West, D. J. & Farrington, D. P (1977) *The Delinquent Way of Life*. London: Heinemann Educational.

Wing, J. (1982) Rehabilitation and management of schizophrenia In *Handbook of Psychiatry, vol 3. Psychoses of Uncertain Aetiology*. (eds J. K. Wing & L. Wing). Cambridge. Cambridge University Press.

World Health Organization (1948) *Official Records of the World Health Organization, No. 2*, pp. 100. Geneva: WHO

—— (1981) *Development of Indicators for Monitoring Progress Towards Health for All by the Year 2000* Geneva: WHO.

Wilson, R (1981) Do health indicators indicate health? *American Journal of Public Health*, **71**, 461

Rachel Jenkins, MD(Cantab), MRCPsych, *Principal Medical Officer, Mental Health Division, Department of Health, Alexander Fleming House, Elephant and Castle, London SE1 6BY, and Honorary Senior Lecturer, Institute of Psychiatry.*

British Journal of Psychiatry (1990), **157**, 515–522

The Functional Psychoses in Afro-Caribbeans

I. HARVEY, M. WILLIAMS, P. McGUFFIN and B. K. TOONE

When 54 Afro-Caribbean and 49 white British consecutive psychotic in-patients were prospectively studied for clinical differences in course of illness and pattern of symptoms, no major differences were found. This does not support the hypothesis that misdiagnosis within the psychoses can explain the higher admission rates of schizophrenia calculated for Afro-Caribbean populations.

Considerable interest has emerged over the apparently high incidence rate of schizophrenia in the Afro-Caribbean population of the UK (Rwegellera, 1977; Bebbington *et al*, 1981; McGovern & Cope, 1987; Cochrane & Bal, 1987; Littlewood & Lipsedge, 1988; Harrison *et al*, 1988). Other studies based on in-patient samples have also noted an excess of functional psychoses among Afro-Caribbean patients (Cochrane, 1977; Carpenter & Brockington, 1980; Dean *et al*, 1981; Cochrane & Bal, 1989; Glover, 1989).

One explanation of these elevated rates is a greater frequency of brief and atypical psychoses in the Afro-Caribbean population, which may be erroneously diagnosed as schizophrenia, particularly since diagnostic reliability can be reduced when assessing someone from another culture (Littlewood & Lipsedge, 1981*a*,*b*; Adebimpe, 1981; Cochrane & Bal, 1987). Rack (1982) suggested that psychogenic psychoses were commoner in the Afro-Caribbean population, being brief disorders in reaction to life stresses with good outcome, but such an excess was not identified by Harrison *et al* (1988, 1989) in their well designed study.

Quite apart from epidemiological issues, a mental disorder with the same phenomenology in various ethnic groups could still exhibit substantial differences in its course and outcome, despite using diagnostic criteria without cultural bias. This would be of equal importance to know in managing individual patients and planning services. If these ethnic differences are common then they should be apparent in a moderately large sample of patients defined by acceptable criteria as having a functional psychosis. If not, then these particular diagnostic issues seem unlikely to explain the elevated rates observed above.

The study reported here is based on a prospective sample of consecutive admissions to one hospital. It provides an opportunity to explore possible clinical differences between Afro-Caribbean and white British patients, without any selection bias after the point of admission. In particular, diagnostic bias can be reduced and examined by including all patients who satisfy broad criteria of psychosis. Although this hospital provides the adult service for an entire catchment area, no attempt was made to identify all patients resident in that area who satisfied these criteria, so that patients seen in other settings were not included.

It is accepted that pathways into psychiatric care differ according to ethnic origin (Rwegellera, 1980; Harrison *et al*, 1984) which may distort hospital data, for example, through greater use of compulsory admissions for Afro-Caribbean patients (Rwegellera, 1980; Littlewood & Lipsedge, 1981*a*). People may be diagnosed psychotic because of their behaviour or experiences that appear bizarre in the majority culture but would not be regarded as evidence of serious mental illness within their own. A consideration of these differential filters before admission is, however, beyond the scope of this study, and such cases of misdiagnosis were excluded if apparent.

Nonetheless, in a clinical study of Afro-Caribbean patients it is important to take into account their specific demographic pattern, as well as social and cultural differences, due to their pattern of immigration into the UK. This increased after the 1952 McCarran Act in the USA prohibited further immigration there, while free migration was permitted to the UK until the Immigration Acts of 1962, 1968 and 1971 progressively restricted further entry. Local population data were therefore required in order to provide such information.

Method

The study was based in the Camberwell Health District, an inner-London area that comprises East Lambeth and South Southwark. The psychiatric unit attached to King's College Hospital takes all adult admissions from East Lambeth with the exception of those admitted to one of the Maudsley Hospital's regional specialist units in the neighbouring district. Population data for East Lambeth were obtained from the 1981 census, using also the projected 1986 population estimates produced by the Office of Population

Censuses and Surveys (OPCS) and the OPCS Small Area Statistics from the 1981 census (OPCS, 1981) for the nine electoral wards that comprise East Lambeth. In 1981 this district was the sixth most socially deprived out of 192.

The patient group consisted of 127 consecutive adult patients admitted with a psychotic illness to this unit over the period March 1986 to March 1987. A further 23 patients, assessed identically in a pilot study during 1985, were included to yield an initial total sample of 150. Patients admitted more than once during the study had only the first admission assessed. Patients over 70 years of age were excluded.

To recruit this sample all psychiatric admissions to the unit were initially screened for a possible psychotic illness using a brief screening check-list that contained the following six items: delusions, hallucinations, catatonic or bizarre behaviour, marked loss of contact with reality, profound lack of insight, and perplexity. Patients who failed to score positively on at least one item were considered not psychotic and excluded; inter-rater reliability on this decision among three of the authors (MW, PMcG and IH) administering the check-list was high (kappa = 0.88, $P = 0.001$). Patients scoring at least one item positively were then interviewed using the Present State Examination (PSE; Wing *et al*, 1974) by a psychiatrist trained in the technique and were included only if they displayed some hallucinatory or delusional phenomena. Patients were not included on the basis of past psychotic phenomena or negative symptoms alone because of the uncertain validity of recorded symptoms and of judgements made largely on the basis of behaviour.

The PSE was usually administered within 24 hours of admission, and in all cases within the first three days; the number of suitable patients who left the unit without being included would have been very few but cannot be quantified in the absence of interview data. Additional information required for diagnoses according to Research Diagnostic Criteria (RDC; Spitzer *et al*, 1977) and DSM–III (American Psychiatric Association, 1980) were collected through patient interview and whatever collateral history was contained in the case notes. An ICD–9 diagnosis was also assigned (World Health Organization, 1978). Judgements about cultural normality were available, if there was doubt, from interview with a relative, which was possible in 54% of Afro-Caribbean patients. All diagnoses took account of past episodes as well as the present one. If a patient was uncommunicative on admission the PSE was repeated at a later date when subjective experiences could be discussed adequately. The Syndrome Checklist (SCL) of the PSE was completed using the case notes for all previous episodes and, together with the other PSE data, entered into the CATEGO program (Wing *et al*, 1974). Eight patients with an organic psychosis were excluded, as were five patients with insufficient diagnostic information. The use of cannabis was not taken as presumptive evidence of an organic psychosis and such patients were therefore retained.

The ethnic group of each parent was recorded as white British, Afro-Caribbean, West African, Indian/Pakistani, or 'other' on the basis of either physical appearance or report by family members. Forty-nine patients were of white British parentage, 54 Afro-Caribbean, nine West African, eight were from other ethnic groups, five were of mixed parentage, and in 12 cases ethnicity could not be reliably ascertained. The first two of these subgroups form the basis of this analysis, giving a sample of 103. The patient's country of birth was also recorded.

Clinical data regarding course of disorder, drug response, previous admissions and other relevant items were acquired from available case notes, and a sociodemographic schedule was completed after interview with an independent informant. Family history of psychiatric illness was constructed from a semistructured interview with the patient and at least one other relative wherever possible. Occupation was coded according to the method of Goldthorpe & Hope (1974). Raters were not blind to the patient's ethnic group.

The total population aged 15–69 for East Lambeth in 1986 was estimated at 57 671 in the OPCS projections. For the Afro-Caribbean population there are no accurate local figures since the 1981 census recorded country of birth but not the ethnic group of each individual, so British-born Afro-Caribbeans cannot be identified. However, an attempt was made to estimate figures for first- and second-generation Afro-Caribbeans separately, using the 1981 census figures on the number of residents in private households subgrouped according to age and birthplace of the head of household. These figures will be a fairly accurate population estimate for the second generation aged 10–19, who were then living with parents born in the West Indies.

TABLE I

Ethnic group and sex of East Lambeth population estimated for 1986 (% of total for that age group in parentheses)

	Male	*Female*	*Total*
Age 15–24 years			
Second-generation Afro-Caribbeans	2112 (13.0)	2349 (14.5)	4461 (27.5)
White British	3911 (24.1)	4016 (24.8)	7927 (48.9)
Other	1894 (11.7)	1929 (11.9)	3823 (23.6)
Total	7917 (48.8)	8294 (51.2)	16211 (100)
Age 45–69 years			
First-generation Afro-Caribbeans	1507 (8.3)	1714 (9.5)	3221 (17.8)
White British	4838 (26.7)	6492 (35.8)	11330 (62.5)
Other	2299 (12.7)	1273 (7.0)	3572 (19.7)
Total	8644 (47.7)	9479 (52.3)	18123 (100)

Nonetheless, it would include a minority born in the West Indies rather than the UK, and exclude some teenagers and young adults immigrating between 1981 and 1986 to rejoin their families. These individuals ($n=4461$) would be aged 15–24 in 1986 and constitute an age group at risk for schizophrenia, particularly in males. This figure could be broken down according to sex using OPCS estimates of the East Lambeth male and female population in 1986. The first half of Table I shows, for this 15–24-year age group, these estimates of the East Lambeth population in 1986.

First-generation Afro-Caribbeans can be identified in those aged 45 or over, but the intervening ages contain both first and second generations, owing to the 15–20-year wave of migration. For this 45–69-year age group a similar extrapolation yields the remainder of Table I.

Results

There were 46 men and 57 women, aged 15–69, with a mean age of 39 years (mode 30). Women were significantly older than men (mean age 43.1 and 34.3 years respectively; Mann–Whitney, $P=0.001$). Of the 54 Afro-Caribbean patients, 14 were born in the UK and 34 in the West Indies; adequate information was unavailable in six. The age and sex distribution for the ethnic groups is given in Fig. 1. Afro-Caribbeans were significantly younger than the white British (mean ages 35.3 and 42.9 years respectively; Mann–Whitney, $P<0.01$), more noticeably in males than females.

Similar proportions in both groups were married (25% and 27%) while 61% of Afro-Caribbeans and 53% of the white British group were single. Fewer Afro-Caribbeans were in first rather than subsequent marriages (10% and 15% respectively), with the converse for white patients (21% and 6% respectively). There were no differences in the number divorced (4% and 6%), separated (8% and 10%), or widowed (2% and 4%).

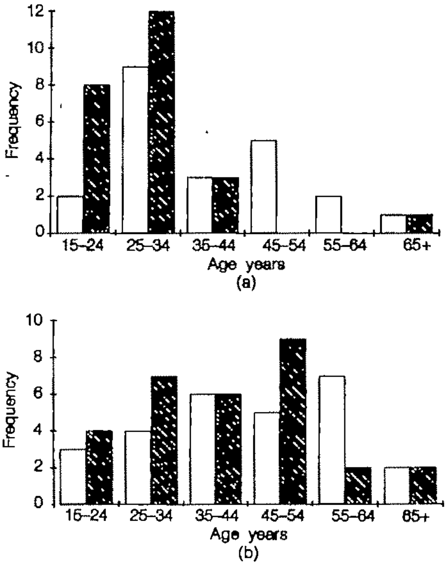


FIG. 1 Age distribution of indigenous British (□) and Afro-Caribbeans (■) in (a) males and (b) females.

TABLE II
Admission rates for any functional psychosis per 100 000 per annum

Age group	*Afro-Caribbeans*	*White British*	*Relative risk for Afro-Caribbeans (95% CI)*
15–24 years	186 ($n=9$)	58 ($n=5$)	3.21 (1.07–9.54)
45–69 years	315 ($n=11$)	155 ($n=19$)	2.03 (0.97–4.29)

Rates of unemployment were higher in Afro-Caribbeans, being 76% and 56% in the two groups (s.e. of % difference = 9.17, $P<0.02$), but previous occupational status was similar (most frequently lower-grade salaried professionals, clerical workers, lower-grade service workers, and semiskilled or unskilled manual workers). Only 4% and 10% in the groups were fully employed, with a further 2% and 6% full-time housewives, 6% and 8% in part-time jobs, 6% and 8% retired, and 6% and 12% registered sick.

Age-specific rates of admission to this hospital for any functional psychosis were calculated using data from the main sample, collected over 13 months, but excluding data for the 23 patients in the earlier pilot study. Rates in each ethnic group for the 15–24-year and 45–69-year age range, using the population estimates in Table I, are shown in Table II. In the Afro-Caribbean population the risk, relative to that of the white British, is seen to be 3.21 and 2.03 respectively, although the 95% confidence interval reaches unity. Admission rates were not calculated separately for each sex, or for schizophrenia alone, as numbers were too small.

Are there psychotic syndromes specific to the Afro-Caribbean group?

(a) In 35 Afro-Caribbean and 35 white British patients, matched for sex and RDC category (19 schizophrenic, 9 schizoaffective, 7 bipolar), a PSE syndrome profile was constructed to examine whether syndromal differences existed which cut across customary classification. Residual schizophrenia and self-neglect were rather less apparent in Afro-Caribbeans, along with depressive phenomena and grandiose/religious delusions. Auditory hallucinations appeared more frequently in Afro-Caribbeans, but overall there was close resemblance between the two groups and none of the above differences were significant. Sixty-nine of those 70 patients had a PSE Index of Definition of at least 5. The mean DAH score (total delusions and hallucinations) derived by CATEGO was identical for the two groups at 5.3.

Diagnosis according to ICD–9, DSM–III or RDC did not show any unexpected differences. Subsequent results relate to the RDC system unless otherwise stated, since DSM–III introduces certain duration criteria, which may

TABLE III

Number of patients according to diagnosis, sex and ethnic group

RDC diagnosis	*Male*			*Female*			*Both sexes*		
	White British	*Afro-Caribbean*	*Total*	*White British*	*Afro-Caribbean*	*Total*	*White British*	*Afro-Caribbean*	*Total*
Schizophrenia	11	17	28	8	11	19	19	28	47
Schizoaffective	3	3	6	6	8	14	9	11	20
Bipolar	2	2	4	7	5	12	9	7	16
Major depression	3	0	3	5	4	9	8	4	12
Unspecified psychosis	3	2	5	1	2	3	4	4	8
Total	22	24	46	27	30	57	49	54	103

prove to differ between groups, and the ICD-9 diagnoses are less precisely defined.

Table III shows the relationship between ethnicity and diagnosis according to sex. It shows an excess of schizophrenia in the Afro-Caribbean group, particularly males. Excluding compulsory admissions does not abolish this trend, as the 15–34-year age group contains 11 Afro-Caribbeans and 7 white-British voluntary patients with schizophrenia.

This difference in admission numbers is only seen in those diagnosed schizophrenic and aged 15–34 (19 Afro-Caribbeans, 10 white British); similar numbers are apparent in other diagnoses, and for schizophrenia in those aged 45–69. However, allowing for the relative population size in the two age groups (the Afro-Caribbean : white-British ratio is 0.56 and 0.28 respectively), it is possible that there is a more diffuse Afro-Caribbean increase in terms of age and diagnosis. The male : female ratio is similarly elevated in both groups for schizophrenia (1.4–1.5).

(b) Comparing individual symptoms in just those patients with a diagnosis of RDC schizophrenia there was close similarity between the two groups. On PSE the Afro-Caribbean patients experienced more neutral and hostile auditory hallucinations (62% v. 39%, s.e. of % difference = 9.6, $P = 0.02$), and more partial delusions of control, while fantastic delusions were less common (5% v. 28%, s.e. of % difference = 7.1, $P = 0.01$). Differences at the PSE syndromal level were minimal. Examination of symptoms throughout their illness, using the SCL, supported the impression that abusive/persecutory auditory hallucinations were commoner in Afro-Caribbeans (67% v. 16%, s.e. of % difference = 8.26, $P < 0.001$), although persecutory delusions were of equal frequency. Negative and affective symptoms were similar in the two groups. In RDC schizophrenia the CATEGO class was NS+ in 42% and 53% respectively.

(c) Those 13 patients (7 Afro-Caribbeans and 6 white British) whose psychotic episodes had never exceeded two weeks in duration were examined to see if a brief psychosis specific to the Afro-Caribbean group was distinguishable. Their symptoms appear in Table IV. Affective and schizophrenic symptoms were simultaneously present in 7 of 13 patients. Using the RDC there were three with schizophrenia, six with schizoaffective and four with bipolar disorders; on DSM-III there were four schizoaffective, four schizophreniform/atypical, and five bipolar patients, while on ICD-9 there were three schizophrenic, two schizoaffective and eight manic–depressive (circular type) patients. There were no ethnic differences in symptoms or diagnoses, apart from delusions of reference which occurred in all seven Afro-Caribbean patients but only in two of six white British patients; this symptom showed a similar pattern in the total sample as well. Of the 13 only four were experiencing their first episode.

Twenty-eight patients with religious delusions were identified, using a positive rating on PSE Item 78 or 79, including 16 Afro-Caribbeans. Only four of these patients had their psychosis resolve within two weeks of its commencement. First-rank symptoms were present in six of 16 women and five of 12 men; seven of those 28 were Afro-Caribbean women without first-rank symptoms, of whom only one was married.

Age at index admission and at first onset of psychosis

Differences between the two groups on these variables were only observed for schizophrenia and bipolar diagnoses. As expected, male schizophrenics had an earlier onset than

TABLE IV

Symptoms in 13 patients with a brief psychosis (duration up to two weeks)

PSE item	*No. of patients with item present*	
Bizarre behaviour	7	
Delusions of reference	9	
Persecutory delusions	5	
Thought insertion	5	
Third-person hallucinations	4	
Affective symptoms	11	
elevated mood		10
irritable mood		8
overactivity		10
pressure of speech		9
distractibility		10
reduced need for sleep		9
initial insomnia		6
early morning wakening		5
loss of appetite		7

females (24.3 v. 34.7 years respectively; Mann–Whitney, $P<0.001$) and a similar pattern was seen for age at admission (29.8 v. 44.6 years respectively; Mann–Whitney, $P<0.005$). This sexual difference in age of onset was 10–11 years in both groups, although greater Afro-Caribbean numbers gave it significance in that group (21.5 years in males v. 32.2 years in females; Mann–Whitney, $P=0.004$) but not in the white British group (28.5 years in males v. 38.3 years in females; NS). For age at admission this difference was still apparent for both Afro-Caribbeans (mean difference of 13.3 years; Mann–Whitney, $P=0.02$) and white British patients (mean difference of 16.7 years; Mann–Whitney, $P=0.07$).

Schizophrenia in Afro-Caribbean patients overall had an earlier mean age of onset by 6.7 years compared with white British patients (25.9 years v. 32.6 years respectively; Mann–Whitney, $P=0.05$), which was reflected in their younger age at admission (33.0 years v. 39.8 years respectively; NS). A similar pattern was seen for each sex separately.

For bipolar disorders, Afro-Caribbean women were younger at admission (mean age 28.3 years v. 41.0 years; Mann–Whitney, $P=0.02$), but the two groups had a mean age of onset that was virtually identical (23.4 and 24.0 years respectively).

Episode onset and course of illness

Over the entire sample of 103 patients there were 16 with a first episode of psychosis, seven of whom were Afro-Caribbean. Twelve of these 16 were first episodes of schizophrenia (five Afro-Caribbeans).

In 35 patients there was no clear period of recovery from a previous psychotic episode, but in the remaining 68 the time between onset of the present episode (i.e. presence of definite psychotic symptoms) and admission was assessed. In 12 this was over six months, while in 35 it was under one month; average duration was similar for each sex and ethnic group. Only six of the 14 patients whose time from onset to admission was one week or less were Afro-Caribbean. There were only 13 patients with psychotic symptoms that lasted in total for two weeks or less, seven of whom were Afro-Caribbean.

Average in-patient stay for the index admission overall was 68.1 days for Afro-Caribbeans and 74.7 days for white British patients. Only in the schizoaffective subgroup was there any difference, with the latter having a mean duration of stay twice as long (30 days v. 66 days respectively; Mann–Whitney, $P=0.04$).

Following admission 31 were still showing psychotic phenomena at the time of discharge while 38 had resolved within two weeks of admission. Duration of delusions and hallucinations exceeded six months at any time in their illness in 39 with no significant ethnic differences in these indices of chronicity, nor in the mean duration of total in-patient stay, which was 11.9 months for Afro-Caribbean and 15.4 months for white British patients. The average number of admissions was 4.6 and 6.4 respectively.

Good symptom recovery between episodes, using a simple dichotomous rating, was estimated to have occurred in 32 of the Afro-Caribbean group and 21 of the white British group; good social recovery was somewhat lower, in 16 and 15 respectively.

Symptom response to neuroleptics was similar across the two groups, with an apparently complete response in 33 patients. However, persistent non-compliance was more frequent in Afro-Caribbean patients ($\chi^2=6.0$, d.f. = 1, $P<0.02$), in 16 of whom the index admission appeared closely related to such non-compliance, whereas this seemed true for only five of the white British group ($\chi^2=7.86$, d.f. = 1, $P<0.01$). Regarding this aspect of non-compliance the two groups were not judged to differ on ratings of poor rapport or lack of insight. Extrapyramidal side-effects (akathisia, dystonia, Parkinsonism and tardive dyskinesia) were equally frequent in both groups. A history of electroconvulsive therapy was less common in Afro-Caribbeans (9/47 v. 15/46; χ^2 NS).

Drug misuse

Illicit drug use was also examined as a possible confounding factor. While 22 of the Afro-Caribbean group acknowledged previous cannabis use only three admitted abusing other drugs; for the white British group 11 admitted abuse of drugs other than cannabis and only three specified cannabis use alone ($\chi^2=19.1$, 2 d.f., $P<0.001$). For 24 patients urine samples were taken on admission and these revealed only cannabis (i.e. negative for amphetamines, opioids, cocaine and barbiturates), which was present in 4 of 16 and 3 of 8 of the two groups respectively. Regular use of cannabis alone in the three months preceding admission was estimated to have occurred in 8 of the former and 3 of the latter. Only one escalated usage during this period, so that any regular use was largely continuance of a pre-existing steady habit.

Drug misuse in the form of a history of deliberate self-poisoning was only seen in one Afro-Caribbean patient but in 11 of the white British group ($\chi^2=7.7$, 1 d.f., $P<0.01$). Similarly a history of alcohol abuse was commoner in the latter (14/49 v. 2/54, $\chi^2=10.3$, d.f. = 1, $P<0.005$).

Family history

Finally, family history of any psychotic disorder in first- or second-degree relatives was examined. A family history of schizophrenia appeared in 6 of 54 of Afro-Caribbeans and 5 of 49 white British patients, of affective psychosis in two of each group, and of unspecified psychosis in four and two respectively. In total 12 and nine respectively had one or more first- or second-degree relatives with a history of psychosis. In the brief psychotic group four of 13 had such a family history.

Discussion

The main finding from this study is that Afro-Caribbean patients admitted to hospital with a functional psychosis are not distinguishable clinically from their white British counterparts. However, it is important to qualify this by the fact that differential rates of referral and admission to hospital have been shown to exist for these two groups (Rwegellera, 1980; Harrison *et al*, 1984). This must preclude any generalisation from this study that such clinical differences do not exist in the general population. Lacking data about the proportion of psychotic patients in each group that do not get admitted, it is not possible to explore this issue further, but it should be borne in mind when evaluating these results.

Sociodemographic differences that were found included younger age at onset of schizophrenia and greater unemployment for Afro-Caribbean patients. However, the local Afro-Caribbean population is noticeably younger than the white British one, and this appears the most likely explanation for their earlier age at onset and admission for schizophrenia, as a lower proportion will have passed through the age range of maximum risk. Burke (1974) found that age of onset for schizophrenia in Jamaica was 25.9 years for males and 29.0 years for females.

Regarding the diagnosis and phenomenology of psychoses in Afro-Caribbean people there is considerable agreement here with the findings of Harrison *et al* (1988, 1989). Their sample comprised 42 Afro-Caribbean patients under the age of 55 with a first-onset functional psychosis, most but not all of whom received in-patient treatment, compared with 89 non-Afro-Caribbean cases from the general population. PSE profiles comparing these two groups for ICD–9 schizophrenia found no major differences to suggest an atypical psychosis phenomenologically. Affective symptoms were actually less frequent in the Afro-Caribbean group, contrary to what one might expect for a brief reactive psychosis. Our findings show a substantial similarity, using both a PSE profile that cuts across diagnostic boundaries and examining symptoms just within the schizophrenic group. In this sample Afro-Caribbean patients with schizophrenia experienced more auditory hallucinations, particularly those with a persecutory or hostile content. Increased auditory hallucinations in schizophrenia have been previously described for Afro-Caribbeans patients living in Camberwell (Chandrasena, 1980). Some evidence suggests that Afro-Caribbeans more commonly report auditory hallucinations in bipolar disorders also (Mukherjee *et al*, 1983).

Furthermore, in terms of duration, Harrison *et al* (1989) found half their Afro-Caribbean patients had had symptoms for at least six months before admission, comparable to the frequency observed here if those with unremitted symptoms from a previous episode were added to those with a definable onset. In that study a similar proportion of Afro-Caribbeans were single (60%), although there were more separated and divorced (25%) and fewer married or cohabiting (14%). In 21% of Afro-Caribbeans a family history of psychosis was elicited, close to the 23% seen in this sample although the latter included second-degree as well as first-degree relatives. They observed illicit drug use, mostly cannabis, in 40% but only in two cases was their psychosis attributed to it, which closely reflected our experience.

Older reports describe an acute atypical psychosis with prominent persecutory ideas and grandiose or religious delusions (Tewfik & Okasha, 1965; Gordon, 1965). However, Gordon noted that the first episode was within two years of immigration in 52% of cases, and only 5% had a family history of psychosis, suggesting a different patient group to that seen here. Littlewood & Lipsedge (1981*b*) also described a brief psychosis presenting typically in married Afro-Caribbean women with religious delusions but an absence of first-rank symptoms.

The present findings suggest that in both ethnic groups psychoses are inherently more pleomorphic if brief rather than sustained, and that there are no features specific to the Afro-Caribbean group, except perhaps more frequent delusions of reference. It may still be the case that a specific brief atypical psychosis does occur in Afro-Caribbeans but it seems unlikely on these results to be very common, and other explanations are possible. Firstly, over the course of two decades or more, the dominant culture has had a pathoplastic effect and delusional content approximates to that culture. Secondly, the changing and mixed phenomenology seen in these brief psychoses means that there is a greater chance of identifying 'atypical' patterns, particularly if the thought content associated with a different culture is novel; Littlewood & Lipsedge (1981*b*) themselves raise both of these possibilities. Unlike these authors we found no excess of brief psychosis in the Afro-Caribbean patients. Patients with religious or supernatural/magical delusions on the PSE were separately identified but again these did not cluster into either an Afro-Caribbean or brief psychotic group, even if first-rank symptoms were excluded. It is important to point out that those few patients who left the unit within three days of admission before inclusion in this study might have involved some with brief psychoses.

Carpenter & Brockington (1980) commented that the apparent increase in schizophrenia among Afro-Caribbean groups might arise from a paranoid psychosis that resembled schizophrenia but lacked first-rank symptoms and was demographically distinguishable. We failed to find support for such an explanation, although other authors agree that first-rank symptoms are less frequent in Afro-Caribbean patients (Chandrasena, 1980; Ndetei & Vadher, 1984*a*) and that persecutory delusions are commoner (Ndetei & Vadher, 1984*b*). Rwegellera (1977) however has argued that the major psychotic syndromes are equally applicable to Afro-Caribbean patients.

The similarities observed across the two ethnic groups extended to features other than phenomenology, such as total length of hospital stay, number of admissions, neuroleptic response and degree of recovery.

The results given here for relative risk in the Afro-Caribbean population are undoubtedly crude estimates, but their function was only intended to provide a link, namely to see if these clinical findings pertained to a sample with some resemblance to the epidemiological studies noted above, since queries about misdiagnosis largely arose from the latter. Nonetheless, some support for these figures is available from Bebbington *et al* (1981), who studied the adjacent population of South Southwark using the Camberwell Register over 1971–78. For the age range 15–64 they found male Afro-Caribbean patients had 2.4 times the admission rate for schizophrenia compared with the white British population, after age correction. Rwegellera (1977) had previously used the same register over 1965–68 and found the inception rate for schizophrenia in the 15–24-year group to be almost nine times higher in Afro-Caribbeans than in the white British, remaining elevated to a lesser degree in older age groups also.

Regarding affective disorders, Leff *et al* (1976) reported higher rates of mania for Afro-Caribbeans in the Camberwell borough, and both Hemsi (1967) and Rwegellera (1977) showed a higher prevalence locally of affective disorders in general. Results from this sample show some support for the position that elevated admission rates for Afro-Caribbeans are not specific to one diagnosis, despite being most obvious for schizophrenia. A similar conclusion is drawn by Glover (1989), since Afro-Caribbean men born after 1952 were at increased risk of admission for affective as well as schizophrenic psychoses. He also found admission rates in young Afro-Caribbean adults of either sex were partly elevated by more frequent re-admissions, but we did not observe such an effect.

In conclusion, the puzzle persists as to which aetiological factors might explain different incidence and admission rates for schizophrenia in the Afro-Caribbean and white British populations. Differential application of that diagnosis is a plausible explanation but has not yet been substantiated.

Acknowledgements

The authors wish to thank the MRC Social Psychiatry Unit, Institute of Psychiatry, for the CATEGO analysis, and Maureen Taylor, Janet Yatak and Clare Sims for secretarial help

References

Adebimpe, V R. (1981) Overview. white norms and psychiatric diagnosis of black patients. *American Journal of Psychiatry*, **138**, 279–285

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) Washington, DC. APA.

Bebbington, P., Hurry, J. & Tennant, C (1981) Psychiatric disorders in selected immigrant groups in Camberwell *Social Psychiatry*, **16**, 43–51.

Burke, A. W. (1974) First admissions and planning in Jamaica. *Social Psychiatry*, **9**, 39–45

Carpenter, L & Brockington, I F. (1980) A study of mental illness in Asians, West Indians and Africans living in Manchester *British Journal of Psychiatry*, **137**, 201–205

Chandrasena, R. D (1980) *Schneider's First Rank Symptoms A Transcultural Evaluation of their Diagnostic Implications and their Prevalence.* MPhil Thesis, Institute of Psychiatry, University of London

Cochrane, R. (1977) Mental illness in immigrants to England and Wales: an analysis of mental hospital admissions, 1971. *Social Psychiatry*, **12**, 25–35.

—— & Bal, S S. (1987) Migration and schizophrenia an examination of five hypotheses. *Social Psychiatry*, **22**, 181–191

—— & —— (1989) Mental hospital admission rates of immigrants to England· a comparison of 1971 and 1981. *Social Psychiatry and Psychiatric Epidemiology*, **24**, 2–11

Dean, G., Walsh, D , Downing, H., *et al* (1981) First admissions of native-born and immigrants to psychiatric hospitals in south-east England 1976. *British Journal of Psychiatry*, **139**, 506–512

Glover, G. R (1989) The pattern of psychiatric admissions of Caribbean-born immigrants in London *Social Psychiatry and Psychiatric Epidemiology*, **24**, 49–56

Goldthorpe, J H. & Hope, K. (1974) *The Social Grading of Occupations – A New Approach and Scale* Oxford Clarendon Press

Gordon, E B. (1965) Mentally ill West Indian immigrants *British Journal of Psychiatry*, **111**, 877–887.

Harrison, G., Ineichen, B , Smith, J., *et al* (1984) Psychiatric hospital admissions in Bristol. II Social and clinical aspects of compulsory admission. *British Journal of Psychiatry*, **145**, 605–611.

——, Owens, D , Holton, A., *et al* (1988) A prospective study of severe mental disorder in Afro-Caribbean patients. *Psychological Medicine*, **18**, 643–657.

——, Holton, A., Neilson, D., *et al* (1989) Severe mental disorder in Afro-Caribbean patients. some social, demographic and service factors *Psychological Medicine*, **19**, 683–696.

Hemsi, L. K (1967) Psychiatric morbidity of West Indian immigrants, a study of first admissions in London. *Social Psychiatry*, **2**, 95–100.

Leff, J P., Fischer, M. & Bertelsen, A (1976) A cross-national epidemiological study of mania *British Journal of Psychiatry*, **129**, 428–442.

Littlewood, R. & Lipsedge, M. (1981*a*) Some social and phenomenological characteristics of psychotic immigrants. *Psychological Medicine*, **11**, 289–302.

—— & —— (1981*b*) Acute psychotic reactions in Caribbean born patients. *Psychological Medicine*, **11**, 303–318.

—— & —— (1988) Psychiatric illness among British Afro-Caribbeans. *British Medical Journal*, **296**, 950–951.

McGovern, D. & Cope, R. V. (1987) First psychiatric admission rates of first and second generation Afro-Caribbeans. *Social Psychiatry*, **22**, 139–149.

Mukherjee, S., Shukla, S., Woodle, J., *et al* (1983) Misdiagnosis of schizophrenia in bipolar patients: a multi-ethnic comparison. *American Journal of Psychiatry*, **140**, 1571–1574.

Ndetti, D. M. & Vadher, A. (1984*a*) A cross cultural study of the frequencies of Schneider's first rank symptoms of schizophrenia. *Acta Psychiatrica Scandinavica*, **70**, 540–544.

—— & —— (1984*b*) Frequency and clinical significance of delusions across cultures. *Acta Psychiatrica Scandinavica*, **70**, 73–76.

Office of Population Censuses and Surveys (1981) *Census 1981*. London: HMSO.

Rack, P. (1982) *Race, Culture and Mental Disorder*. London: Tavistock.

Rwegellera, G. G. C. (1977) Psychiatric morbidity among West Africans and West Indians living in London. *Psychological Medicine*, **7**, 317–329.

—— (1980) Differential use of psychiatric services by West Indians, West Africans and English in London. *British Journal of Psychiatry*, **137**, 428–432.

Spitzer, R. L., Endicott, J. & Robins, E. (1977) *Research Diagnostic Criteria (RDC) for a Selected Group of Functional Disorders* (3rd edn). New York: Biometrics Research Division, New York State Psychiatric Institute.

Tewfik, G. I. & Okasha, A. (1965) Psychosis and immigration. *Postgraduate Medical Journal*, **41**, 603–612.

Wing, J. K , Cooper, J. E. & Sartorius, N. (1974) *Measurement and Classification of Psychiatric Symptoms*. London: Cambridge University Press.

World Health Organization (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

*Ian Harvey, MSc, MRCPsych, *Honorary Lecturer, Institute of Psychiatry, DeCrespigny Park, London SE5 8AF*; Maureen Williams, MA, DipPsych, *Principal Clinical Psychologist, King's College Hospital, Denmark Hill, London SE5 8RX*; Peter McGuffin, PhD, FRCPsych, FRCP, *Professor of Psychiatry, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN*; Brian K. Toone, MPhil, FRCPsych, FRCP, *Consultant Psychiatrist, King's College Hospital, Denmark Hill, London SE5 8RX*

*Correspondence

PROZAC

ecause depressed patients still have a life to lea

Prozac, like the tricyclics, is highly effective in the treatment depression.[1,2]

But that's where the similarity ends.

As a highly specific 5HT reuptake inhibitor, Prozac has significant effect on the patient's psychomotor performance.[3,4]

Anticholinergic side effects are less frequent and severe n those of tricyclic drugs too.[2]

So depressed patients can get on with a normal daily life thout being dazed by daytime drowsiness.[3,4]

Other side effects are usually mild and manageable, th few patients feeling the need to abandon treatment.[5]

It is also reassuring to know that Prozac has nimal effect on the heart,[6] and that the dangers lowing overdose are low.[7]

Prozac controls the patient's depression, whilst leaving them to resume and cope with their normal everyday activities.

All from just one 20mg capsule a day.

PROZAC
fluoxetine

THE OUT AND ABOUT ANTIDEPRESSANT

Abbreviated Prescribing Information.

▼ Special reporting to CSM required.

'PROZAC' (Fluoxetine Hydrochloride).

Presentation Capsules containing 20mg fluoxetine hydrochloride. **Uses** Treatment of the symptoms of depressive illness. Prozac has been shown to decrease significantly binge-eating and purging activity. **Dosage and Administration** (For full information, see data sheet). For oral administration to adults only. *Depression:* A dose of 20mg/day is recommended. Because of the long elimination half-lives of the parent drug and its major metabolite, changes in dose will not be fully reflected in plasma for several weeks. *Bulimia:* A dose of 60mg/day is recommended. The maximum daily dose should not exceed 80mg for any indication. *The elderly:* The maximum total daily dose should not exceed 60mg. *Children:* Not recommended. *Patients with renal and/or hepatic dysfunction:* See 'Contra-indications' and 'Precautions' sections. **Contra-indications** Hypersensitivity to fluoxetine. Prozac should not be administered to patients with severe renal failure (GFR < 10ml/min). *Usage in nursing mothers:* Prozac should not be prescribed to nursing mothers. **Warnings** *Monoamine oxidase inhibitors:* At least 14 days should elapse between discontinuation of an MAOI and initiation of treatment with Prozac. At least five weeks should elapse between discontinuation of Prozac and initiation of therapy with an MAOI. While a causal relationship to fluoxetine has not been established, death has occurred following initiation of MAOI therapy shortly after discontinuation of Prozac. *Rash and possibly allergic events:* Prozac should be discontinued upon appearance of rash or of other possibly allergic phenomena for which an alternative aetiology cannot be identified. Systemic events, possibly related to vasculitis, have developed. Although rare, this may be serious, involving lung, kidney or liver Death has occurred. Serum sickness, anaphylaxis and pulmonary events, including inflammatory processes and/or fibrosis, have been reported. *Usage in pregnancy:* The safety of Prozac in human pregnancy has not been established. **Precautions** Prozac should be avoided in patients with unstable epilepsy and it should be discontinued in any patient who develops seizures. A lower dose of Prozac, eg alternate day dosing, is recommended in patients with significant hepatic dysfunction or mild to moderate renal failure (GFR 10-50ml/min). Caution is advisable when Prozac is used in patients with acute cardiac disease. Prozac may cause weight loss which may be undesirable in underweight depressed patients. In diabetics, fluoxetine may alter glycaemic control. There is little clinical experience of the concurrent administration of fluoxetine with ECT or lithium therapy (See 'Drug interactions'). There have been case reports of prolonged seizures in patients on fluoxetine receiving ECT treatment. Rare reports of altered platelet function and/or abnormal laboratory values, and several reports of abnormal bleeding. *Drug interactions:* Greater than 2-fold increases of previously stable plasma levels of other antidepressants have been observed when Prozac has been administered in combination. Agitation, restlessness and gastrointestinal distress have been reported in five patients receiving fluoxetine in combination with tryptophan. Increased (with lithium toxicity) or decreased lithium levels have been reported. Lithium levels should be monitored. Pharmacokinetic data suggest that the half-life of diazepam may be prolonged in some patients. *For further information, see data sheet.* **Side-effects** *Depression:* The following treatment-emergent adverse events were observed during placebo controlled clinical trials at a frequency of one per cent or greater and at a significantly higher incidence than placebo (P value ≤ 0.05):– Asthenia, fever, nausea, diarrhoea, mouth dryness, appetite loss, dyspepsia, headache, nervousness, insomnia, drowsiness, anxiety, tremor, dizziness, fatigue, decreased libido, pharyngitis, dyspnoea excessive sweating, (rash, see 'Warnings'), sexual dysfunction. *Bulimia:* Using the same criteria; insomnia, nausea, asthenia, tremor, sweating, decreased libido. The more common events listed above that caused discontinuation include nausea, headache, nervousness, insomnia, anxiety, dizziness and asthenia. Other events that have been reported include vomiting and convulsions. During pre-marketing testing hypomania or mania occurred in approximately 1 per cent of fluoxetine treated patients. Elevated serum transaminase values and/or depressed leucocyte counts without accompanying symptoms occurred infrequently in patients given fluoxetine. Voluntary reports of adverse events temporally associated with fluoxetine, that have been received since market introduction and which may have no causal relationship with the drug, include: cerebral vascular accident, confusion, dyskinesia, ecchymoses, gastro-intestinal haemorrhage, hyperprolactinaemia, pancreatitis, suicidal ideation, thrombocytopenia, thrombocytopenic purpura, vaginal bleeding after drug withdrawl and violent behaviours. Hyponatraemia (including serum sodium below 110mmol/l) has been rarely reported. This appears to be reversible upon discontinuation. *Overdosage:* As of December 1987, there have been 2 deaths in patients who took overdoses of fluoxetine in combination with other drugs (maprotiline, codeine, temazepam). Except for these deaths, all other 36 overdose cases which involved fluoxetine either alone or in combination with other drugs and/or alcohol recovered without complications. One patient who reportedly took 3000mg of fluoxetine experienced 2 grand mal seizures that remitted spontaneously. Since introduction, a single death, attributed to overdose of fluoxetine alone, has been reported. **Legal Category** POM **Product Licence Number** 0006/0195 **Basic NHS Cost** £27.44 per pack of 28 capsules. **Date of Preparation** July 1990.

References 1. Feighner JP. *J Clin Psychiatry* 1985; **46**: 369-372. 2. Stark P, Hardison CD. *J Clin Psychiatry* 1985; **46** (3, Sec 2): 53-58. 3. Schaffler K. *Br J Clin Pract* 1986; **40** (Suppl. 46): 28-33. 4. Hindmarch I. *Human Psychopharmacol Bull* 1987; **2**: 177-183. 5. Cooper GL. *Brit J Psych* 1988; **153** (Suppl. 3): 77-86. 6. Fisch C. *J Clin Psychiatry* 1985; **46** (3, Sec 2): 42-44. 7. Data on file. Dista Products Limited.

Full Prescribing Information is available from:

DISTA

Dista Products Limited, Dextra Court, Chapel Hill, Basingstoke,
Hampshire RG21 2SY.
Telephone: Basingstoke (0256) 52011.

'PROZAC' is a Dista trade mark. PZ205

PROZAC
fluoxetine

THE OUT AND ABOUT ANTIDEPRESSANT

Part-time Retired Consultant Psychiatrist for Private Mental Health Clinic, Surfers Paradise, Queensland, Australia

This is a fee-for-service clinic with two psychiatrists. Would suit retired NHS consultant interested in business holiday for one or more years doing part-time work, golf, etc. Salary by hours worked. Must have MRC Psychiatry before 1980 for Queensland registration. Beachside cottage and car available.

Apply:
Dr Michael Craft,
P.O. Box 254, Mermaid Beach,
Queensland, Australia, 4218.

GASKELL Royal College of Psychiatrists

Health Services Planning and Research

Contributions from Psychiatric Case Registers

Edited by J. K. Wing
£7.50, 136 pp.
ISBN 0 902241 30 3

British Journal of Psychiatry (1990), 157, 523–528

Eating Behaviour and Attitudes to Weight and Shape in British Women from Three Ethnic Groups

BRIDGET DOLAN, J. HUBERT LACEY and CHRIS EVANS

Attitudes towards eating, weight and shape were examined in 479 Caucasian, Afro-Caribbean and Asian British women. The Asian women were found to have significantly more disordered eating attitudes than the Caucasian women, but no difference was found between the three groups in their concern with their body weight and shape. However, while in the Caucasian group disordered eating attitudes were significantly positively correlated with feelings of anxiety and depression, this was not true in the other two groups. Although the concerns of British Afro-Caribbean and Asian women are similar to those of the Caucasian women, there may be ethnic differences in the relationship between feelings about eating, weight and shape and mood.

Bulimia, an eating disorder which occurs in women of normal body weight, has an estimated prevalence ranging from 2% of the general British female population to 13% of a student population (Cooper *et al*, 1984, 1987*a*). Lesser eating problems such as occasional binge eating, self-induced vomiting, laxative abuse and extreme dieting have been found in up to a quarter of British women attending a general practice (Fairburn & Cooper, 1983).

The absence of black or Asian women in epidemiological studies of eating-disordered populations has been the subject of comment (Bruch, 1965). Psychiatric case-register studies in both Britain and the United States have shown the incidence of anorexia nervosa in non-white women to be markedly less than that for Caucasians (Kendell *et al*, 1973; Jones *et al*, 1980). In America, surveys have shown the prevalence of eating disorders to be much lower in black than in white college students (Nevo, 1985; Gray *et al*, 1987) and very few cases of anorexia nervosa in blacks have been reported (Pumareiga *et al*, 1984; Silber, 1984; Robinson & Andersen, 1985). Furthermore, to our knowledge, only four case reports of black women with bulimia attending clinics in the United States exist (Lawlor & Rand, 1985; Hsu, 1987).

In Britain, the first report of anorexia in Afro-Caribbean girls did not appear until 1985 (Thomas & Szmukler, 1985; a further study from the same centre being described by Holden & Robinson, 1988). The authors recently reported the first five cases of bulimia in non-white British women (Lacey & Dolan, 1988).

Mumford & Whitehouse (1988*a*), however, surveyed 204 Asian and 355 Caucasian schoolgirls in Bradford, England, using the Eating Attitudes Test (EAT: Garner *et al*, 1983) and the Body Shape Questionnaire (BSQ; Cooper *et al*, 1987*b*) and found that there was no difference in the BSQ scores of the two ethnic groups but that the mean EAT score of the Asian girls was significantly *higher* than that of the Caucasian group ($P < 0.001$). Seven Asian and three Caucasian girls received a diagnosis of bulimia nervosa (DSM–III–R; American Psychiatric Association, 1987). The one-year prevalence of this condition was 3.4% and 0.8% respectively, the difference being statistically significant. These findings are at variance with the clinical reports that suggest white women make up the overwhelming majority of referrals to specialist treatment centres. An earlier study from our own catchment-area clinic (Lacey & Dolan, 1988) noted that the ratio of non-white:white referrals was 1:55, at most one-tenth of the ratio of non-white:white women in the catchment area.

Several alternative explanations have been proposed for the small numbers of black and Asian women referred to eating disorders clinics. It is sometimes suggested that eating disorders rarely occur in non-Caucasian women, or that, given similar distressing circumstances and emotional disturbance, black women will present with different neurotic disorders. It has also been implied that black women are not under the same cultural pressure as white Western women to value slimness as desirable. Certainly, in several non-Western cultures, fatness and obesity symbolise beauty (Nasser, 1988). However, these attitudes to weight may be modified by the acculturative pressure upon non-white British women (Furnham & Alibhai, 1983). This discrepancy may also reflect a lack of referral of ethnic minority groups to the National Health Service (NHS). The use of the NHS psychiatric services by minority populations remains a subject of debate (London, 1986). Although there are several studies of psychiatric illness in British ethnic minorities, many studies have concentrated upon in-patient admission rates and their findings are contradictory (Bagley, 1971; Cochrane, 1977; Carpenter & Brockington, 1980; Dean *et al*, 1981). Existing research has not disentangled the many possible variables affecting this issue.

This study measured and compared eating attitudes and behaviour as well as feelings about weight and shape in three ethnic groups in the light of ethnic attendance rates in the eating disorders clinic. A secondary aim was to explore possible mediating effects of anxiety and depression.

Method

A self-report questionnaire, compiled for this study, was divided into three sections. The first section included a demographic questionnaire which recorded information about the subject's age, marital and employment status and her current height, weight and minimum and maximum adult weights. Ethnicity was judged from information about the place of birth of the subject and her parents. Information about the subject's first language and that of her parents was also requested.

The second section consisted of the EAT-26 and the BSQ. The former was used to quantify disordered attitudes towards eating and food and to address a broad range of symptoms of anorexia nervosa in three subscales (dieting, bulimia and food preoccupation). The EAT has been used as a screening device for the clinical disorder and found to have satisfactory reliability and validity (Garner & Garfinkel, 1979). Questions are presented in Likert form and possible scores on the EAT-26 range from 0 to 78, the clinical cut-off point being 20. The BSQ is a 34-item questionnaire measuring female concerns with body weight and shape. It particularly considers the behavioural antecedents and consequences of 'feeling fat' in addition to general concerns with body image. The BSQ has been validated in clinical and non-clinical populations. Questions are presented on a six-point Likert scale, giving a possible range of 34–204. No clinical cut-off point has been described.

The third section comprised the 14-item Hospital Anxiety and Depression Scale (HAD; Zigmond & Snaith, 1983), designed for use in general hospital out-patient populations; it avoids the physical symptoms of anxiety and depression, concentrating more on psychological symptoms. Its validity and reliability have also been shown (Zigmond & Snaith, 1983).

The survey population consisted of all women attending a family planning and well-woman clinic in a single health centre in south-west London over three months. While in the waiting area, all women attending the clinic were asked by the first author (BD) if they would complete a questionnaire about their attitudes to eating and dieting. It was emphasised that participation was voluntary and results would be completely confidential. The questionnaire was completed by 517 women (97%); six women refused, giving no reason (three Caucasian, two Afro-Caribbean, one Asian), and nine women said they had difficulty in reading English (six Asian, two Oriental, one African).

The total sample was classified into seven ethnic groups: Caucasian; Afro-Caribbean (including Guyanan); Asian (including Indian, Pakistani, Sri Lankan and East African Asian); African; mixed race; Mauritian; and Oriental.

Data were entered into a microcomputer and analysed using SPSS (Norusis, 1988). Conventional parametric tests were used as the Levene test revealed no significant heteroscedasticity. Duncan's *post-hoc* range tests and 95% confidence intervals (CI) for the mean of samples are presented where appropriate (Langman, 1986). Principal-component and internal-reliability analyses (Cronbach, 1951) were conducted within ethnic groups to test for cultural specificity in the measures (Mumford & Whitehouse, 1988*b*; Bhugra & King, 1988).

Results

The total sample consisted of 365 (70.6%) Caucasian, 71 (13.7%) Afro-Caribbean, and 43 (8.3%) Asian women. Because of the small numbers, the 38 (7.4%) women of other ethnic groups (15 African, 16 mixed race, six Mauritian and one Oriental) were excluded from any further analysis, leaving a final sample of 479 women.

The mean age of the total sample was 28.2 years, the mean age of the Asian group being the highest at 31.7 years. Analysis of variance showed a significant group effect (*F* ratio 5.16, *F* probability <0.001), the Asian group being significantly older than the Caucasian and Afro-Caribbean women (Duncan range test).

The majority of women had never been married (58%). However, when marital status was considered with ethnicity, it was found that for the Caucasian and Afro-Caribbean women, approximately a third were married and two-thirds were single, while for the Asian women, this ratio was reversed. There was a significant difference in the parity of women in the three groups ($P<0.001$, Kruskal-Wallis). However, there was no difference in parity of the groups if only those women who had ever been married were considered.

Weights and heights were transformed to Quetelet indices (W/H2), but population weight norms for different British ethnic groups are unfortunately not available. The Afro-Caribbean women had a higher mean Quetelet index (24.2, s.d. 4.4) than the Caucasian (21.9, s.d. 3.1) or the Asian women (23.1, s.d. 4.2) ($P<0.05$).

The overall mean EAT-26 score was 8.89 (s.d. 9.2). Analysis of variance showed a significant group effect (*F* ratio = 3.02, *F* probability = 0.049). A Duncan's range test revealed that the Asian women scored significantly higher than the Caucasian group ($P<0.05$; Table I). For the BSQ, the overall sample mean was 84.2 (s.d. 35.3). Analysis of variance showed no significant group effects. The standard deviations were large in all groups (Table I).

EAT and BSQ scores may be related to weight and age. To allow for the possible interaction of these variables, an analysis of covariance was performed covarying out the effect of age and Quetelet index. For the EAT, a significant group difference remained after covariance (ANCOVA *F*

TABLE I
Mean (s.d.) EAT and BSQ scores (with 95% confidence intervals and percentage above cut-off score (20) for EAT only)

	EAT scores				*BSQ scores*		
Ethnic group	n	*Mean (s.d.)*	*CI 95%*	*% above cut-off score*	n	*Mean (s.d.)*	*CI 95%*
Caucasian	353	8.53 (9.5)	7.54–9.52	8.5%	344	85.3 (35.2)	81.63–89.08
Afro-Caribbean	57	8.79 (6.8)	6.96–10.6	7.0%	56	79.3 (35.2)	69.88–88.76
Asian	38	12.39 (9.7)	9.21–15.5	16.0%	34	80.7 (37.6)	67.62–93.85
Total sample	448	8.89 (9.2)	8.03– 9.7		434	84.2 (35.3)	80.88–87.55

(2,423) = 3.4, $P<0.001$). For the BSQ scores, analysis of covariance with age and Quetelet index also showed a significant effect ($F(2,412)=4.12$, $P<0.02$).

For the depression subscale of the HAD, analysis of variance showed a significant group effect (F ratio 7.39, F probability <0.001). Duncan's range test showed that the Asian group scored significantly higher than the Caucasian group ($P<0.05$). The anxiety subscale of the HAD produced an unexpectedly high score in all groups; the total sample mean was higher than the clinical cut-off point of eight, and 51% of the total sample scored eight or above. There was no significant group effect by analysis of variance (Table II).

Correlation matrices for EAT score, BSQ score and HAD subscales were calculated in the three groups (Table III). For each group there was a significant positive correlation between the EAT and BSQ scores and between the anxiety and depression subscales of the HAD. For the Caucasian group, the EAT and BSQ scores also correlated positively with both HAD scales. The Afro-Caribbean group's BSQ scores were positively correlated with the two HAD subscales while for the Asian group there was no correlation between either of the two eating scales and either of the two mood subscales.

Factor and reliability analyses

The Cronbach's alpha values (Table III) are all sufficient to justify use of the scales in all samples, with the possible exception of the value for the HAD depression subscale in the Asian group. Exploratory factor analysis was performed on the EAT, BSQ and HAD questionnaires. The EAT was found to have a first factor accounting for 32.4%, 20.6% and 22.5% of the variance in the Caucasian, Afro-Caribbean and Asian groups respectively. The loadings on this factor were similar in all groups, loading patterns on the smaller factors showing less consistency across groups. Factor analysis of the BSQ showed that, for each group, one main factor accounted for the majority of the sample variance. This was 53.5% for the Caucasian group, 52.3% for the Afro-Caribbean group and 50.3% for the Asian group. The questions loading on this factor were similar for each group. For each group the only question loading less than 0.4 on this first factor was number 26, 'Have you ever vomited to feel thinner?'

Despite the low reliability value for the depression scale in the Asian group, the factor analyses of the HAD showed a comparable first factor for each group accounting for 38%, 33.7% and 27.4% of the variance. This first factor corresponded closely to the anxiety measure with the addition of one question from the depression scale ('I feel as if I am slowed down') also loaded above 0.4 on this factor in all three groups. The second factor for each group seemed to be a depression factor, accounting for 9.8%, 13.6% and 14.9% of the variance. Further details of all these analyses will be reported elsewhere.

Discussion

The results show that abnormal eating attitudes and concern with body weight and shape are found in all groups of women, regardless of their ethnic background. As found by Mumford & Whitehouse (1988*a*), EAT socres were highest in the Asian group and there was no ethnic difference in BSQ scores. For the total sample, the mean EAT score was similar to that of Garner *et al* (1983) of 9.9 (s.d. 9.2) and

TABLE II
Mean (s.d.) HAD scores (with 95% confidence intervals)

Ethnic group	*Depression score Mean (s.d.)*	*CI 95%*	*Anxiety score Mean (s.d.)*	*CI 95%*
Caucasian	3.81 (3.06)	3.5–4.1	8.30 (4.12)	7.86–8.74
Afro-Caribbean	4.79 (3.32)	3.9–5.7	7.34 (4.25)	6.25–8.43
Asian	5.30 (3.06)	4.5–6.7	8.11 (3.88)	6.76–9.47
Total sample	4.09 (3.15)	3.7–4.3	8.15 (4.12)	7.70–8.54

TABLE III

Correlation matrices for EAT, BSQ and HAD (anxiety and depression) scores (Cronbach's alpha values in parentheses on the leading diagonal)

	EAT score	*BSQ score*	*HAD anxiety score*	*HAD depression score*
Caucasian group				
EAT score	*(0.87)*			
BSQ score	0.63*	*(0.97)*		
HAD anxiety score	0.38*	0.52*	*(0.84)*	
HAD depression score	0.26*	0.44*	0.63*	*(0.75)*
Afro-Caribbean group				
EAT score	*(0.71)*			
BSQ score	0.55*	*(0.97)*		
HAD anxiety score	0.28	0.37**	*(0.83)*	
HAD depression score	0.15	0.60*	0.45*	*(0.72)*
Asian group				
EAT score	*(0.87)*			
BSQ score	0.56*	*(0.97)*		
HAD anxiety score	0.05	0.26	*(0.71)*	
HAD depression score	0.04	0.23	0.48***	*(0.58)*

*$P<0.001$, **$P<0.006$, ***$P<0.01$

the mean BSQ score was similar to that found by Cooper *et al* (1987*b*) in the initial validation study of the BSQ (81.5, s.d. 28.4). Factor analysis has shown that similar first factors exist in all three ethnic groups for the EAT, BSQ and HAD questionnaires which supports the comparison of total scores across the groups.

As well as the differences in the main dependent variables, we also found differences between the groups in this sample on age, Quetelet index, parity and marital status. However, an analysis of covariance for the EAT partialling out age and Quetelet index still showed a significant difference between groups.

The main intention of this study was to explore whether the ethnic difference in referral rate at the eating disorders clinic was matched by similar differences in mean scores on questionnaire measures of eating attitudes in women attending another local clinic. The unequivocal finding is that there were sizeable ethnic differences in these mean scores but that these are opposite in direction to the ethnic differences in attendance rate at the eating disorders clinic. However, it must be emphasised that the EAT is not a clinical diagnostic instrument.

Our sample is not representative of the general population since the women were attenders at a family-planning clinic. McAvoy & Raza (1988) discuss cultural differences in usage of family-planning services which, given the selected nature of our sample, could mediate the group differences in eating attitudes. However, even if these factors do mediate group differences in eating attitudes this should not detract from the finding that some non-white women demonstrate high EAT scores, yet are not represented in referrals to the eating-disorders clinic.

The self-definition of illness is an important factor in determining who reaches treatment for a disorder. In our own clinic referral is always secondary, 73% of referrals being from general practitioners (GPs) the remainder being from physicians, surgeons and other professionals. For 83% of referrals, the woman has herself approached the GP with her eating problem. Despite the secondary referral, therefore, the bulimia is largely self-defined in the majority of cases. A study of GP consultation in Oxford showed that there was no difference between Asian and Caucasian women's rates of consultation (Brewin, 1980), although there was a much lower rate of referral for psychiatric treatment for Asians in that area. Similarly, Murray & Williams (1986) found no differences in the rate of GP consultations by Asian and white women in west London. If we can assume that the discrepancy in referral of black and Asian women is not because of non-presentation to GP *per se*, it may be because they do not present to their GP complaining of an eating disorder. Maybe only Caucasian women think that self-starvation or binge eating are worrisome and in such a way that doctors might be of some help. It may also be that the attitudes and culture of health-care professionals themselves influence the likelihood of a disorder being identified. Ways of showing distress differ between cultures, as does the labelling of behaviour as aberrant. Perhaps doctors do not recognise, or do not look for, eating disorders in non-white women.

Although we have shown a similar level of disordered eating attitudes in different ethnic groups, there may be a difference in the way the groups construe such attitudes and how these may relate to or directly affect mood. In the clinic, a depressed mood and low self-esteem are usually an integral part of the presentation of an eating disorder. This association between disordered eating attitudes, low mood and high anxiety was only significant among Caucasian women in our sample. Although the different sizes of the groups indicate a need for caution in interpreting this result, it is in some way compatible with the differences in clinic referral rate (i.e. although non-white women have disordered attitudes to eating and symptoms of disorder, these are not strongly associated with depression and anxiety). It is likely that some degree of emotional distress about the disordered eating is necessary to precipitate a request for help or definition of the behaviour as a problem.

The aetiology of eating disorders has been linked to the emphasis upon slimness as desirable for women in Western societies, and it has been implied that black and Asian women are not under this same cultural pressure (Hsu, 1987). Certainly, disordered eating attitudes and overconcern with weight and shape are ubiquitous and culturally acceptable in white Western women (Nylander, 1971). However, the present study has shown that these attitudes and concerns are not limited to Caucasian British women. Both Afro-Caribbean and Asian women showed similar body shape concerns.

Few studies of attitudes to female body shape in different ethnic groups have been reported. Furnham & Alibhai (1980) considered the preference for female body shape of Kenyan Asian women, British women and Kenyan Asian immigrants to Britain. They found that the Kenyan Asian women rated fatter figures more favourably and smaller figures less favourably than did the British women. The immigrant women, however, were even more extreme than the British women in their perceptions, reacting most positively to thinner shapes.

Other evidence for the cultural determination of morbid concern with body weight is found in studies within subcultures of Caucasian populations such as those exacting extreme standards of thinness (models, dancers, racing jockeys) in which the prevalence of eating disorder is high (Garner & Garfinkel, 1980; King & Mezey 1987). Perhaps the Western sociocultural ideals of female body shape and the construing of self-esteem in terms of external characteristics may become more evident in non-white women through successive acculturation of different generations.

Further studies could consider generational differences in eating attitudes and correlations with anxiety and depression in different ethnic groups. A complete exploration of all the possible effects and interactions between these factors and confounding variables would require very large samples which can probably only be achieved through multicentre co-operation.

Acknowledgement

This work was supported by a grant from the Special Trustees of St George's Hospital.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R). Washington, DC: APA.

BAGLEY, C. (1971) Mental illness in immigrant minorities in London. *Journal of Biosocial Science*, **3**, 449–459.

BHUGRA, D. & KING, M. (1988) Bulimia nervosa amongst Asian schoolgirls (letter). *British Medical Journal*, **297**, 1198.

BREWIN, C. (1980) Explaining the lower rates of psychiatric treatment among Asian immigrants to the United Kingdom: a preliminary study. *Social Psychiatry*, **15**, 17–19.

BRUCH H. (1965) The psychiatric differential diagnosis of anorexia nervosa. Anorexia Nervosa Symposium, April 1965, Gottingen.

CARPENTER, L. & BROCKINGTON, I. F. (1980) A study of mental illness in Asians, West Indians and Africans living in Manchester *British Journal of Psychiatry*, **137**, 201–205.

COCHRANE, R. (1977) Mental illness in immigrants to England and Wales: an analysis of mental hospital admissions, 1971. *Social Psychiatry*, **12**, 25–35.

COOPER, P. J., WATERMAN, G. C. & FAIRBURN, C. G. (1984) Women with eating problems: a community survey. *British Journal of Clinical Psychology*, **23**, 45–52.

——, CHARNOCK, D. J. & TAYLOR, M. J. (1987*a*) The prevalence of bulimia nervosa: a replication study. *British Journal of Psychiatry*, **151**, 684–686.

——, TAYLOR, M. J., COOPER, Z , *et al* (1987*b*) The development and validation of the body shape questionnaire. *International Journal of Eating Disorders*, **6**, 485–494.

CRONBACH, L. J. (1951) Coefficient alpha and the internal structure of tests. *Psychometrika*, **16**, 297–334.

DEAN, G., WALSH, D., DOWNING, H., *et al* (1981) First admissions of native born and immigrants to psychiatric hospitals in South East England 1976. *British Journal of Psychiatry*, **139**, 506–512.

FAIRBURN, C. G. & COOPER, P. J. (1983) The epidemiology of bulimia nervosa: two community studies. *International Journal of Eating Disorders*, **2**, 61–67.

FURNHAM, A. & ALIBHAI, N. (1983) Cross-cultural differences in the perception of female body shapes. *Psychological Medicine*, **13**, 829–837.

GARNER, D. M. & GARFINKEL, P. E. (1979) The Eating Attitudes Test: an index of the symptoms of anorexia nervosa. *Psychological Medicine*, **9**, 273–279.

—— & —— (1980) Sociocultural factors in the development of anorexia nervosa. *Psychological Medicine*, **10**, 647–656.

——, OLMSTEAD, M. P., BOHR, Y., *et al* (1983) The Eating Attitudes Test: psychometric features and clinical correlates. *Psychological Medicine*, **12**, 871–878.

GRAY, J. J., FORD, K., KELLY, L. M. (1987) The prevalence of bulimia in a black college population. *International Journal of Eating Disorders*, **6**, 733–740.

HOLDEN, N. L. & ROBINSON, P. H. (1988) Anorexia nervosa and bulimia nervosa in British blacks. *British Journal of Psychiatry*, **152**, 544–549.

Hsu, L. K. G. (1987) Are the eating disorders becoming more common in blacks? *International Journal of Eating Disorders*, **6**, 113–125.

Jones, D. J., Fox, M. M., Babigian, H. M., *et al* (1980) The epidemiology of anorexia nervosa in Munro County, New York 1960–1976. *Psychosomatic Medicine*, **42**, 551–558.

Kendell, R. E., Hall, D. J., Hailey, A., *et al* (1973) The epidemiology of anorexia nervosa. *Psychological Medicine*, **3**, 200–203.

King, M. & Mezey, G. (1987) Eating behaviour of male racing jockeys. *Psychological Medicine*, **17**, 249–253.

Lacey, J. H. & Dolan, B. M. (1988) Bulimia in British blacks and Asians: a catchment area study. *British Journal of Psychiatry*, **152**, 73–77.

Langman, M. J. S. (1986) Towards estimation and confidence intervals. *British Medical Journal*, **292**, 716.

Lawlor, B. A. & Rand, C. S. W. (1985) Bulimia nervosa in a black woman (letter). *American Journal of Psychiatry*, **142**, 1514.

London, M. (1986) Mental illness among immigrant minorities in the United Kingdom. *British Journal of Psychiatry*, 149, 265–273.

McAvoy, B. R. & Raza, R. (1988) Asian women. *Health Trends*, **20**, 11–17.

Mumford, D. B. & Whitehouse, A. M. (1988*a*) Increased prevalence of bulimia nervosa amongst Asian Schoolgirls. *British Medical Journal*, **297**, 718.

—— & —— (1988*b*) Bulimia nervosa amongst Asian schoolgirls (letter). *British Medical Journal*, **297**, 1404.

Murray, J. & Williams, P. (1986) Self-reported illness and general practice consultations in Asian-born and British-born residents of west London. *Social Psychiatry*, **21**, 139–145.

Nasser, M. (1988) Culture and weight consciousness. *Journal of Psychosomatic Research*, **32**, 573–577.

Nevo, S. (1985) Bulimic symptoms: prevalence and ethnic differences among college women. *International Journal of Eating Disorders*, **4**, 151–168.

Norusis, M. J. (1988) *SPSS/PC+ V2.0*. Chicago: SPSS Inc.

Nylander, I. (1971) The feeling of being fat and dieting in a school population. *Acta Socio-Medica Scandanavia*, **1**, 17–26.

Pumariega, A. J , Edwards, P. & Mitchell, C. B. (1984) Anorexia nervosa in black adolescents. *Journal of the American Academy of Child Psychiatry*, **23**, 111–114.

Robinson, P. & Andersen, A. (1985) Anorexia nervosa in American blacks. *Journal of Psychosomatic Research*, **19**, 183–188.

Silber, T. (1984) Anorexia nervosa in black adolescents. *Journal of the National Medical Association*, **76**, 29–32.

Thomas, J. P. & Szmukler, G. S. (1985) Anorexia nervosa in patients of Afro-Caribbean extraction. *British Journal of Psychiatry*, **146**, 653–656.

Zigmond, A. S. & Snaith, R. P. (1983) The hospital anxiety and depression scale. *Acta Psychiatra Scandanavia*, **67**, 361–370.

*Bridget Dolan, PhD, *Research Fellow*; J. Hubert Lacey, MD, FRCPsych, *Reader*, Chris Evans BA, MRCPsych, *Clinical Research Fellow in Forensic Psychiatry: Bulimia Clinic and Research Team, Department of Psychiatry, St George's Hospital Medical School, London SW17 0RE*

*Correspondence

British Journal of Psychiatry (1990), **157**, 529–532

Suicide in England and Wales and in Scotland
An Examination of Divergent Trends

IAIN K. CROMBIE

The suicide rates in England and Wales and Scotland converged during the late 1960s, and from 1970 were higher in Scotland. The convergence occurred because of differences in the falls in rates of those aged over 45 years and not because of differences in frequency and decline of coal gas suicide. Male suicide increased more rapidly in Scotland during the 1970s. In England and Wales overall female suicide rates fell during 1958–76 whereas in Scotland there was no clear trend. Suicide by car exhaust increased similarly in both countries, but that by hanging and drug poisoning increased more rapidly in Scotland.

The suicide rate in Scotland has been lower than that in England and Wales for most of this century (Kreitman, 1972; World Health Organization, 1982). Although Barraclough (1972) suggested that this could be a result of differing procedures for the registration of suicide (Barraclough, 1972), a study of case records from both countries indicated that this was not so (Ross & Kreitman, 1975).

Recent studies of trends in suicide in England and Wales (McClure, 1987) and Scotland (McLoone & Crombie, 1987) have shown that the rates among men in both countries have increased while there have been small decreases in those among women. This paper compares the suicide rates in the two countries between 1958 and 1986 to see how their relative positions have been affected by these recent trends.

Method

Data on population size and numbers of suicides for the period 1958–86 were obtained from the Annual Reports of the Registrar General (Scotland) and from the Registrar General's statistical reviews of England and Wales (latterly the Office of Population Censuses and Surveys). Suicide rates were expressed per 100 000 population, and trends in rates were investigated using three-point moving averages.

Results

The trends in suicide rates among men and women in England and Wales and Scotland are shown in Fig. 1. In both sexes the suicide rates in Scotland were much lower than in England and Wales in the late 1950s. The rates converged during the 1960s and by the early 1970s in both men and women were higher in Scotland. The trends in suicide for men and women in England and Wales are dominated by the large falls in the 1960s which were associated with the substitution of natural gas for coal gas. Since then, the rates for men have shown a moderate rise, while for women they have decreased slightly. The trends in Scotland were different. Among men the fall in the rates

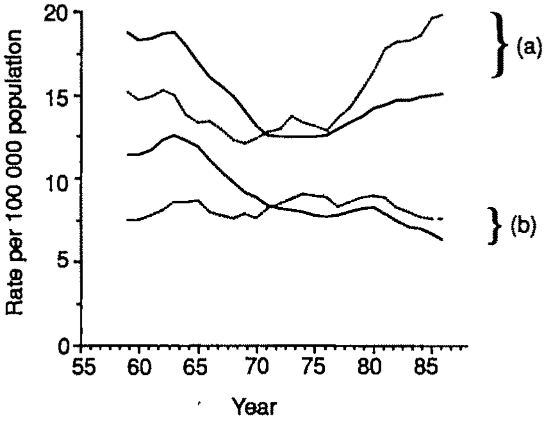


FIG. 1. Trends in suicide rates per 100 000 population among (a) men and (b) women aged 15+ years in Scotland (·····) and England and Wales (——), 1958–1986 (data expressed as three-point moving averages).

TABLE I

Suicide rates per 100 000 population and the percentage of suicides using coal gas in those aged 15–44 years and 45+ years in Scotland and in England and Wales 1958–60

	Men		*Women*	
	Scotland	*England & Wales*	*Scotland*	*England & Wales*
Rate per 100 000 pop.				
15–44 years	9.4	9.9	4.9	5.5
45+ years	22.4	29.1	10.3	16.9
% suicides using coal gas				
15–44 years	51.1%	46.5%	45.5%	54.5%
45+ years	45.1%	45.7%	50.5%	55.0%

TABLE II

Observed and predicted changes in suicide rates in Scotland and England and Wales from 1958–60 to 1974–76 following the withdrawal of coal gas (a positive value indicates an increase in the suicide rate, a negative one, a fall in the rate)

	Changes in suicide rates			
	Scotland		*England & Wales*	
	Observed	*Predicted*[1]	*Observed*	*Predicted*[1]
Men				
15–44 years	+1.6	−4.8	−0.7	−4.6
45+ years	−6.6	−10.1	−12.7	−13.3
Women				
15–44 years	+1.8	−2.5	−0.7	−3.0
45+ years	+0.9	−5.2	−6.3	−9.3

1. Change in rate predicted solely from the withdrawal of coal gas.

during the 1960s was smaller and among women it was barely discernible. Subsequently, the rates for men have shown a very large increase, while those for women have shown little overall change.

Kreitman (1972) showed that the difference in rates between the countries were large among those aged over 45 years, but small among those of younger age groups. Separate analysis for the ages 15–44 years and 45+ years from 1958 to 1960 confirms this (Table I). In both men and women at this time, the rates were much higher in England and Wales at ages above 45 years, but at younger ages there was little difference.

In the 1950s, 45–55% of all suicides in both age groups in Scotland and England and Wales used domestic gas as a method of suicide (Table I). After about 1960 the two countries showed similar decreases in the use of this method for suicide, and by 1974 it had virtually ceased occurring (data not shown).

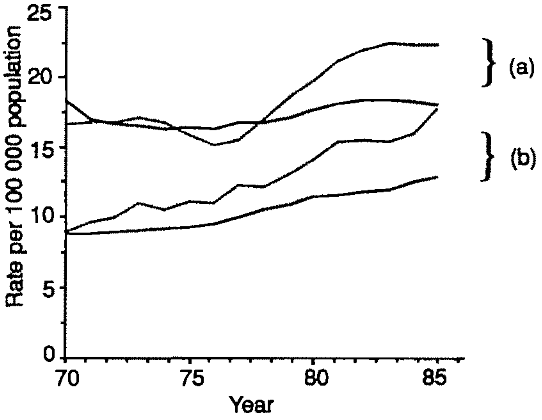


FIG. 2. Trends in suicide rates per 100 000 population among men aged (a) 45+ years and (b) 15–44 years in Scotland (· · · · · ·) and England and Wales (——), 1970–1986 (data expressed as three-point moving averages).

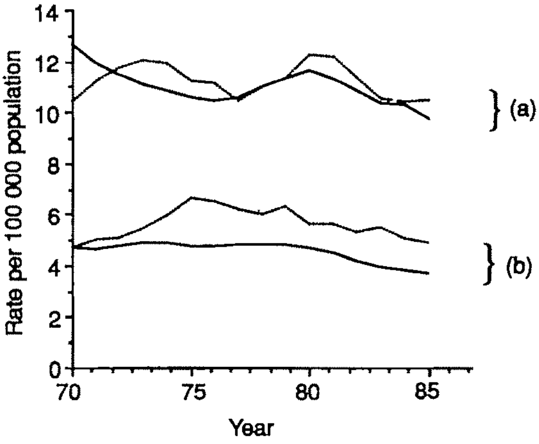


FIG. 3. Trends in suicide rates per 100 000 population among women aged (a) 45+ years and (b) 15–44 years in Scotland (· · · · ·) and England and Wales (——), 1970–1986 (data expressed as three-point moving averages).

The observed falls in suicide rates over the period 1958–76 are compared with the falls that would have been predicted solely from the withdrawal of coal gas (Table II). In general, observed falls were smaller than predicted ones, and only among men and women over 45 years in England and Wales, and among older men in Scotland, were substantial falls in suicide rates observed. Small falls were seen among those aged 15–44 years in England and Wales. In contrast, in Scotland there were small increases among the younger men and women and older women over this period.

Since the falls in overall suicide rates were lower than those expected, there must have been increases in suicide by methods other than coal gas. Fig. 2 shows that among younger men, the small increase in rates observed 1958–76 continued to 1985, but with a more rapid increase in Scotland. Among older men, a rapid increase in rates occurred in Scotland in the late 1970s, but the rise was more modest in England and Wales. Figure 3 shows that, by contrast, there was little change in overall suicide among women over this period.

The methods of suicide which have shown large increases in frequency are shown in Table III. Among Scottish men in both age groups there were large increases in suicide by solid and liquid substances (mainly medical drugs), hanging and suffocation, and other gases (mainly car exhaust). In England and Wales, although the increases in suicide by car exhaust were similar to those in Scotland, the increases for hanging and drugs were smaller, especially at older ages. The fall in suicides by other methods among those aged over 45 years in England and Wales occurred largely in the categories of drowning and firearms (data not shown). For women, increases in suicide by drugs and hanging and suffocation account for the higher Scottish rates. In neither country has suicide by car exhaust become common among women.

TABLE III

Suicide rates per 100 000 population by method for men and women aged 15–45 years and 45+ years in Scotland and England and Wales for 1958–62 and 1982–86

Method	*Men*				*Women*			
	Scotland		*England & Wales*		*Scotland*		*England & Wales*	
	1958–62	*1982–86*	*1958–62*	*1982–86*	*1958–62*	*1982–86*	*1958–62*	*1982–86*
Aged 15–45 years								
Coal gas	4.7	0.1	4.7	0.0	2.3	0.0	2.9	0.0
Solids & liquid substances	1.1	3.4	1.4	2.0	1.4	2.7	1.5	1.7
Other gases	0.2	3.2	0.5	3.9	0.0	0.4	0.0	0.4
Hanging, strangling & suffocation	1.4	5.1	1.3	3.4	0.2	0.8	0.3	0.7
Other methods	2.2	4.7	2.6	2.9	0.9	1.5	1.0	1.0
Aged 45+ years								
Coal gas	10.4	0.0	12.8	0.0	5.2	0.0	9.2	0.0
Solids & liquid substances	1.9	4.3	3.6	3.6	2.5	4.8	4.5	4.9
Other gases	0.2	3.1	0.6	3.4	0.0	0.2	0.1	0.4
Hanging, strangling & suffocation	3.2	7.1	4.4	6.3	0.5	1.4	1.1	2.4
Other methods	6.5	8.0	7.5	4.6	2.6	4.2	2.7	2.2

Discussion

In the 19th century, Durkheim (1897) noted that, with regard to the international differences in suicide rates, the relative position of countries remained constant over time. For most of this century this was true of Scotland and England and Wales, but this study has shown that since about 1970 their positions have reversed. These two countries, because of their proximity, have many common features and might therefore have been expected to have similar suicide rates. In studies using data on suicide before 1970, the concern was to explain why the Scottish rates were so much lower. For example, Kreitman (1972) found that population characteristics could not explain the differences between the countries. The concern now is why the rates have undergone divergent trends so that in Scotland the rates are higher than those in England and Wales by 30% and 20% among men and women respectively.

The divergent trends between the countries during the 1960s were followed in the 1970s by divergent trends between the sexes in both England and Wales (McClure, 1987) and Scotland (McLoone & Crombie, 1987). These trends were not, however, an artefact of changes in the allocation of deaths to the category 'undetermined whether or not purposely inflicted' (McClure, 1987; McLoone & Crombie, 1987).

One important finding of the present study is the different ways in which the withdrawal of coal gas affected the overall suicide rates. In England and Wales, falls in rates were seen in all age and sex groups, although they were more substantial among those aged over 45 years. In Scotland, a fall in rate only occurred among men aged over 45 years, the other groups experiencing rises where falls were predicted. These trends reflect changes in rates of suicide by other methods.

One important determinant of change in suicide rates is availability of method (Oliver & Hetzel, 1973; Farmer & Rhode, 1980), but it is unlikely that changes in availability of means can explain the divergent trends between Scotland and England and Wales. Among men in Scotland, suicide by hanging and strangulation, a method under no restriction, showed the largest increases. Suicide by drugs also showed large increases in men and women in Scotland although both countries have the same health services.

Durkheim (1897) emphasised the importance of social causes of suicide, and social change may explain trends in suicide rates (World Health Organization, 1982). It is well known that Scotland has experienced considerably more social deprivation than England and Wales (Holterman, 1975; Carstairs & Morris, 1989), but the amount of actual data on trends in this and other social factors is limited.

Change in unemployment rates is one obvious candidate, although in his comprehensive review of suicide and unemployment, Platt (1984) concluded that the association was inconsistent. Further detailed analysis of trends in suicide and unemployment within Scotland (Crombie, 1989) and among industrial countries (Crombie, 1990) provide no support for the association. Another possible factor is the extension of the alcohol licensing hours which occurred in Scotland in 1976. However, surveys before and immediately after this change and in 1984 showed no increase in consumption over this period (OPCS, 1985).

The main conclusion from the present study is that a complex but unknown set of social changes in Scotland and England and Wales have resulted in divergent trends in suicide. As McClure (1987) has pointed out, "hopes for a decrease in the rates must rely on dealing with the causes of suicide", for which a greater understanding is needed.

References

BARRACLOUGH, B. M. (1972) Are the Scottish and English suicide rates really different? *British Journal of Psychiatry*, **120**, 267–273.

CARSTAIRS, V. & MORRIS, R. (1989) Deprivation: explaining differences in mortality between Scotland and England and Wales. *British Medical Journal*, **299**, 886–889

CROMBIE, I. K. (1989) Trends in suicide and unemployment in Scotland, 1976–86. *British Medical Journal*, **298**, 782–784.

—— (1990) Can changes in unemployment rates explain the recent changes in suicide rates in developed countries. *International Journal of Epidemiology*, **19**, 412–416.

DURKHEIM, E. (1897) *Le Suicide* (trans. J. A. Spaulding & C. Smith, 1952). London: Routledge & Kegan Paul.

FARMER, R. & RHODE, J. (1980) Effect of availability and acceptability of lethal instruments on suicide mortality. *Acta Psychiatrica Scandinavica*, **62**, 436–446.

HOLTERMAN, S. (1975) Areas of deprivation in Great Britain: an analysis of 1971 census data. *Social Trends*, **6**, 33–47.

KREITMAN, N. (1972) Suicide in Scotland in comparison with England and Wales. *British Journal of Psychiatry*, **120**, 83–87.

MCCLURE, G. M. G. (1987) Suicide in England and Wales, 1975–1984. *British Journal of Psychiatry*, **150**, 309–314.

MCLOONE, P. & CROMBIE, I. K. (1987) Trends in suicide in Scotland 1974–84: an increasing problem. *British Medical Journal*, **295**, 629–631.

OFFICE OF POPULATION CENSUSES AND SURVEYS (1958–86) The Registrar General's statistical reviews of England and Wales. London: OPCS.

—— (1985) Drinking and attitudes to licensing in Scotland. *OPCS Monitor*, 55 85/Z.

OLIVER, R. G. & HETZEL, B. S. (1973) An analysis of recent trends in suicide rates in Australia. *International Journal of Epidemiology*, **2**, 91–101.

PLATT, S. (1984) Unemployment and suicidal behaviour: a review of the literature. *Social Science and Medicine*, **19**, 93–115.

REGISTRAR GENERAL (Scotland) (1958–86) *Annual Reports*. Edinburgh: HMSO.

ROSS, O. & KREITMAN, N. (1975) A further investigation of differences in suicide rates of England and Wales and of Scotland. *British Journal of Psychiatry*, **127**, 575–582.

WORLD HEALTH ORGANIZATION (1982) *Changing Patterns in Suicide Behaviour*. Copenhagen: WHO.

Ian K. Crombie, BSc, MSc, PhD, *Senior Lecturer, Department of Community Medicine, Ninewells Hospital and Medical School, Dundee DD1 9SY*

s Droleptan* is a major tranquilliser for rapidly calming the manic agitated patient. **Dosage** Dosage should be individually determined and titrated according to response t
account age, symptomatology, and previous response to other neuroleptics. In adults, 5-15mg i.v.; up to 10mg i.m.; 5-20mg p.o.; repeated every 4-6 hours i.m./i.v. or 4-8 l
Children 0.5 — 1mg/day im or oral, adjusted according to response. **Use in the Elderly** In the elderly or debilitated use half the normal starting dose. **Contra-indica**
atose states, severe depression. **Precautions** Not recommended during breast feeding. Caution advised in liver disease, renal failure, Parkinson's disease, epileps
litions predisposing to epilepsy or convulsions. May increase effects of CNS depressant drugs. May antagonise action of adrenaline and other sympathomimetics. May enh
effects of methyldopa, may impair anti-Parkinson effects of levodopa. Increased dosage of anticonvulsants may be required. Gradual withdrawal is advisable. **Use in Pregn**
y in human pregnancy has not been established. **Side Effects** In common with all neuroleptics, major side effects including, extrapyramidal symptoms, tardive dyski
ional effects e.g. hyperprolactinaemia, hypotension (particularly in elderly) have been reported. Abnormalities of liver function, epileptic fits and blood dyscrasias have
rted rarely. Possibility of neuroleptic malignant syndrome should be borne in mind. Minor side effects — see data sheet. **Overdosage** Supportive treatment and anti-Park
s as required. **Presentation** Yellow, uncoated, scored tablets marked JANSSEN on one side and D/10 on the reverse, containing 10mg droperidol. Clear colourless aqu
tion in 2ml ampoules containing 5mg/ml droperidol. Clear colourless odourless liquid containing 1mg/ml droperidol. **Product Licence**
. Droleptan tablets 0242/5004R, injection 0242/5003R, liquid 0242/0080. **Basic NHS Cost** Amps (5mg/ml) 2ml × 10 £8.38, Liquid
/ml) 100ml £4.17, 500ml £19.81, Tablets 10mg × 50 £11.42 (correct at time of preparation March 1990). *Trademark. JPL501/90.

JANSSE

GASKELL Royal College of Psychiatrists

Lectures on the History of Psychiatry

The Squibb Series

Edited by R. M. Murray & T. H. Turner **£10, 236 pp, ISBN 0 902241 32 X**

"Thus in psychiatry he was entirely self-taught. . . ." Alexander Walk, Chapter 2, Medico-psychologists, Maudsley and The Maudsley

"It was Jones who conceived the idea of the famous Komittee whose main function was to defend Freud from having to face any further public criticisms. They were to function secretly, and Freud presented each of the members with an antique Greek intaglio . . . mounted in gold rings. Jones was the only member of the Komittee to lose his ring." Thomas Gruffydd Davies, Chapter 4, 'Truth is a point of view': an account of the life of Dr Ernest Jones

"Insane ear seems to have been commonest in general paralytics, and its disappearance, from the literature at least, coincided with the decline of general paralysis. Yet general paralysis continued to be a common disorder for decades after insane ear had apparently gone." Edward Hare, Chapter 6, Old familiar faces: some aspects of the asylum era in Britain

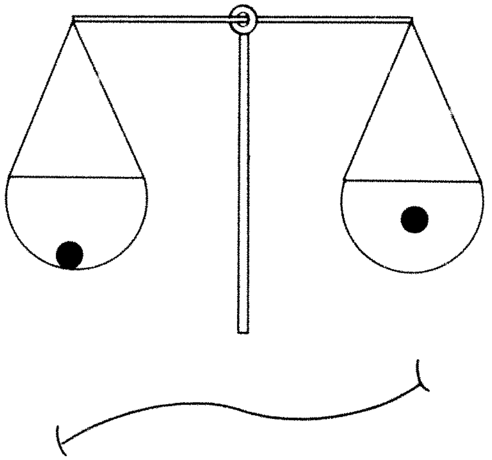
"This English disease, affecting, he insisted, as many as one-third of all people of quality in England, was the result of high living, prosperity, and progress unique on such a wide scale in early 18th-century England. Cheyne flattered his patients: only those of quick and lively dispositions and good breeding suffered from the nerves." William F. Bynum, Chapter 8, The nervous patient in 18th- and 19th-century Britain: the psychiatric origins of British neurology

"Abnormal childhood sexuality was an equally strong clue to the presence of 'chronic sepsis and toxemia . . . [and] in a number of cases the colon has been resected with improvement in the individual'." Andrew Scull, Chapter 10, Desperate remedies: a Gothic tale of madness and modern medicine

"Claude Bernard . . . opposed . . . the view that averages or indeed statistics could ever lead to exact science." German Berrios, Chapter 12, Memory and the cognitive paradigm of dementia during the 19th century: a conceptual history

---

GASKELL Royal College of Psychiatrists



Child Psychiatry and the Law

Edited by Dora Black, Stephen Wolkind & Jean Harris Hendriks
£10, 192 pages, ISBN 0 902241 31 1

British Journal of Psychiatry (1990), **157**, 533–538

Suicide in Cork and Ireland

MICHAEL J. KELLEHER and MAURA DALY

The official suicide rate in Ireland increased markedly between 1970 and 1985. This is a genuine increase in suicide and not an apparent one due to previous deficiencies in recording practices Possible reasons for this change include an increase in *'anomie'* shown by a rise in the rates of crime, illegitimacy and admissions to hospital for alcoholism, a decline in social cohesion revealed by a fall in the marriage rate and a rise in the number of separated couples, and an increase in unemployment.

The official suicide rates in Ireland, which for most of the century were among the lowest in the world, began to rise in the early 1970s and showed an increase per 100 000 population in almost every year from 1.75 in 1970 to 8.0 in 1985. There were many who suggested that the official suicide rates in Ireland were never as low as officially portrayed and that the rise after 1970 simply reflected the more accurate recording of the cause of death. Clarke-Finnegan & Fahy (1983) and McCarthy & Walsh (1975) noted considerable under-reporting of suicide by coroners in Ireland. However, Walsh (1976) concluded that even though the 'true' suicide rate in Ireland should be four times the official rate, it was still remarkably low by international standards. Support for this came from Sainsbury & Barraclough (1968) who found that the suicide rates of immigrant groups in the USA ranked in the same order as those of their country of origin, and that the rate among those living in the USA who were born in Ireland was very low when compared with other immigrant groups. Similar results were found in Australia (Lester, 1972).

Many factors probably contribute to an individual act of suicide. Physical and mental illness, alcoholism, personality structure, social isolation, life events and long-term difficulties have all been implicated in the past. Sociologists, however, claim that analysis of the reasons for individual suicide cannot give an adequate explanation of a country's suicide rate. They point to the marked differences in the suicide rates between countries and between groups within countries and suggest that the number of suicides among individuals experiencing adverse life circumstances depends on group attitudes, which can be influenced by social, economic and environmental changes. Durkheim (1897) held that the suicide rate is related to the degree of integration in society. As the degree of integration, regulation, support and control in society declines, so the suicide rate rises. He also suggested that especially in times of economic upheaval, there were many individuals who could see no legitimate way to satisfy their desires and, in consequence, their behaviour became aimless and antisocial. In this state of society, which Durkheim calls *'anomie'*, the suicide rate would rise.

Sainsbury *et al* (1978) tested Durkheim's concepts of integration and *anomie*. They chose 15 social variables, some of which indicated the extent of *anomie* and family cohesion in society, and others of which measured the socioeconomic status, the degree of alienation of the aged, and the status of women. They correlated the mean values of these variables with the suicide rate of 18 different countries. What clearly emerged was the extent to which changing social conditions affect national trends in suicide.

The present study was carried out because of clinical concern for the marked increase in the number of suicides, particularly those by younger men, which occurred in Cork in the 1970s and 1980s. The main aims of the study were as follows.

(a) To determine whether the increase in the suicide rate in Ireland was real or was due to a change in the practice of recording deaths. It was argued that if the increasing suicide rate was due to a change in recording practice, then as the suicide rate increased, the rate of "deaths undetermined, whether accidentally or purposely inflicted" as well as that of certain accidents such as drowning and poisoning would fall.

(b) To determine, from an examination of public records, whether those factors previously associated with *anomie* and family cohesion, or those which were a measure of socio-economic status, had shown any changes in the years between 1970 and 1985.

(c) To investigate the background to all the suicides in Cork City and County known to the police and coroners, and to note their personal characteristics, social environment and the presence or absence of mental illness.

Method

The data were compiled from a number of sources. The Central Statistics Office (CSO; 1970–85*a*), which publishes statistics annually referring to two or three years previously, gave the numbers of suicides in Ireland, classified by sex and age according to nine possible methods of death (E950–E959) for each year. It also gave a corresponding category (E980–E989) under the heading "deaths undetermined, whether accidentally or purposely inflicted". Furthermore, rates from rural and urban populations from the same source were compared.

To measure the changes in the level of *anomie* in Irish society between 1970 and 1985, we used the illegitimacy rates (CSO, 1970–85*b*), the crime rates (Garda Siochana Stationary Office, 1985), and the number of admissions to hospital for alcoholism (Health Research Board, 1971–85). To estimate the degree of 'integration' in Irish Society we used the marriage rate and the proportion of the population under 15 years (CSO; 1970–85*c*). The numbers of 'married but separated' were taken from the estimates of the Labour Force Survey (CSO, 1984–86) which involved a structured interview with 4.5% of the population, and the 1986 census (CSO, 1986) was used to identify the number of emigrants from Ireland. The unemployment rate was used as a measure of socioeconomic status of the country. The information on unemployment was obtained from Eurostat (1982) for 1971–80 and from the *Irish Statistical Bulletin* (CSO, 1986) for 1981–85. Details on unemployment relevant to Cork were taken from the Cork Corporation's (1984–86) *Cork Economic Monitor*.

The coroners of Cork City and County were interviewed and individual cases of suicide recorded during 1980–85 were examined and their personal characteristics noted. These included the age, sex and previous psychiatric history, if known, as well as the social circumstances including marital and employment status and whether they lived in an urban or rural setting at the time of their deaths.

Results

The suicide rate in Ireland increased in almost every year from 1.75 per 100 000 population in 1970 to 8 per 100 000 population in 1985. Figure 1 compares the suicide rate in Ireland with the rates of 'open verdicts', accidental drownings and accidental poisonings. There is a sharp rise in the suicide rate, but the sum of the rates of the other three verdicts does not vary very much until the 1980s when they show a decrease of 24 per 1 000 000 population: in 1970 the sum of the rates of these three verdicts was 69 per 1 000 000 and in 1985 it was 45 per 1 000 000.

Figure 2 compares the changes over 15 years in the age-specific male suicide rates in Ireland and England and Wales. There were large increases in the rates in all age groups among men in Ireland between 1970 and 1985. The increase in the rates of younger men is marked and appears to be part of a European trend (Sainsbury *et al*, 1978). In England and Wales the rates in different age groups did

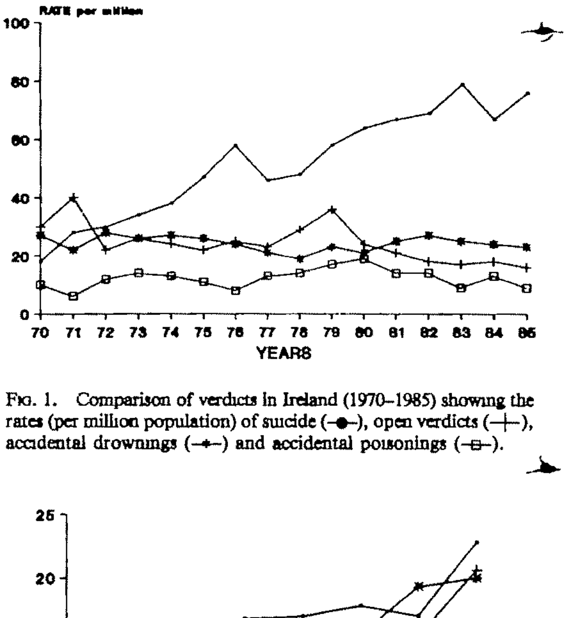


FIG. 1. Comparison of verdicts in Ireland (1970–1985) showing the rates (per million population) of suicide (-●-), open verdicts (-+-), accidental drownings (-*-) and accidental poisonings (-⊟-).

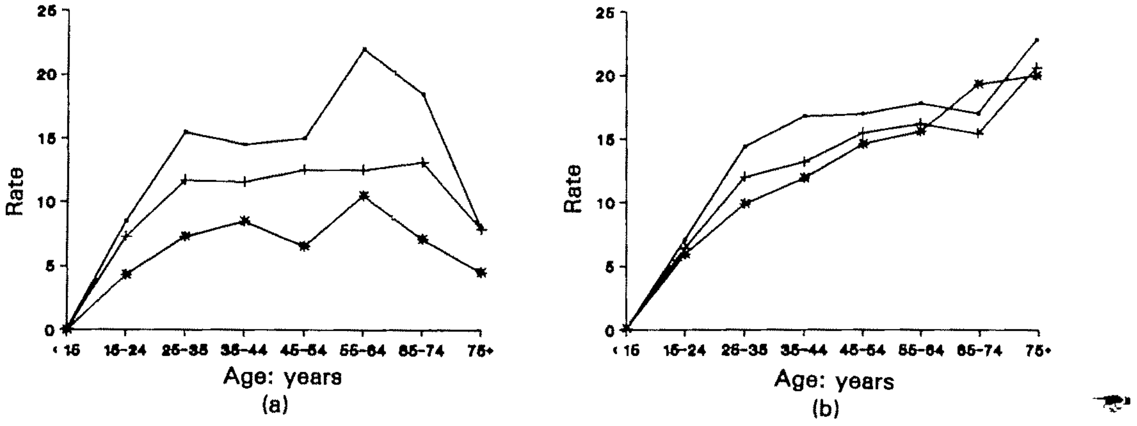


FIG. 2. Comparison of age-specific male suicide rates (per 100 000 population) in (a) Ireland and (b) England and Wales in three time periods, 1970–75 (-●-), 1976–80) (-+-) and 1981–85 (-*-).

not change much in the 1970s but there was a trend in the 1980s for an increase in the 24–64-year age groups. This trend has increased since 1982, and McClure (1987) demonstrated a 37% increase in this age group between 1975 and 1984. The female suicide rate also showed marked increases in all age groups, the greatest increase occurring in the 44–64-year age groups.

Hanging (E953) and drowning (E954) were the principal methods of suicide used by men during the period of the study and accounted for much of the increase in the suicide rates. Suicide by poisoning with solid and liquid substances (E950) and to a lesser extent suicide by shooting (E955) also showed increase over the period. Other methods of suicide including inhaling exhaust fumes were used only sporadically. Drowning (E954) and poisoning (E950) accounted for almost all the increase in the female suicide rate. There was sporadic use of the other methods but none of these showed any sustained increase.

The rural suicide rate exceeded the urban rate for the entire period of the study. Following 1978, the rural rate rose much more steeply than the urban rate so that in 1985, the former was 13 per 100 000 population and the latter 4.5 per 100 000 population. However, the suicide rates for the two largest cities also showed very marked and sustained increases from 1977 onwards (Fig. 3). By 1985, the rate for these cities was in excess of 10 per 100 000 population compared with the remaining urban rate of 2.5.

Figure 4 shows the changes over the period in those variables chosen to measure the level of *anomie* in society. The illegitimacy rate increased in every single year from 5.7 per 1000 population in 1970 to 12 in 1985. The number of admissions to hospital for alcoholism increased from 3073 in 1970 (10 per 1000 population) to a peak of 7345 (21 per 1000 population) in 1981. The number of indictable crimes doubled over the period 1975–85.

Indices of family cohesion indicated a decline in this. The marriage rate fell from 14.8 in 1971 to 10 in 1985 (Fig. 4). The percentage of the population under 15 years fell from 31.2 in 1971 to 30.3 in 1982. There is no divorce in Ireland but the Labour Force Survey has shown an increase in the number of 'married but separated' people every year since 1983 when this report was commenced. The estimate was 21 000 in 1983 and by 1986 had reached 28 900. The 1986 census (CSO, 1986) took note of emigration for the first time and reported that 75 000 had emigrated between 1980 and 1986.

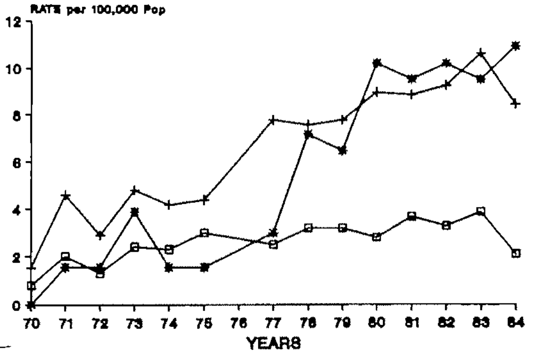


FIG. 3 Comparison of suicide rate (per 100 000 population) in the two largest cities (Dublin (–+–) and Cork (–✱–) and remaining urban area (–⊟–) (1970–84) (no data available for 1976).

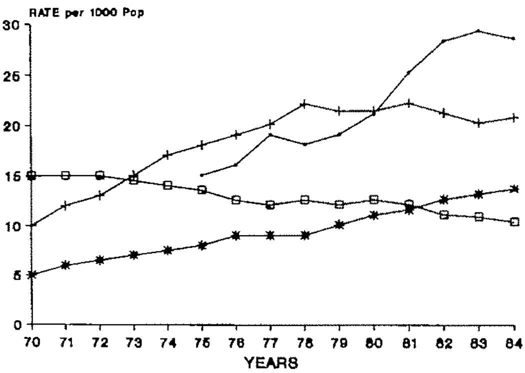


FIG. 4 Indices of *anomie* – marriage (–⊟–), illegitimacy (–✱–), alcohol admissions (–+–) and crime (–✱–) (1970–84).

TABLE I

Suicide rate per 100 000 population and percentage of workforce unemployed (1971–85) in Ireland and Cork

	1971–73	*1974–76*	*1977–79*	*1980–82*	*1983–85*
Ireland					
Unemployed: % of workforce	5	6.7	7.5	10.0	15.0
Suicide rate per 100 000 population	3	4.7	5.1	6.4	7.2
Cork[1]					
Unemployed: % of workforce	—	—	—	12.0	19.3
Suicide rate per 100 000 population	—	—	—	10.3	11.2

1. No values for unemployment rates in Cork available 1971–79.

With regard to socioeconomic status, Table I shows the relationship between the suicide rate and the percentage of the workforce unemployed. For Ireland as a whole and for Cork, the suicide rate rose in parallel with the unemployment rate.

The Cork study

From 1970 to 1975, the suicide rates (per 100 000 population) of Cork City and Cork County were 1.78 and 4.0 respectively compared with a national rate of 3.3. For the period 1980–85 the rate (per 100 000 population) of Cork City was 10.76, that of Cork County was 8.2, and the national rate was 6.9. Of the 207 people who committed suicide in Cork in the years 1980–1985, 88 came from the city (60 men and 28 women) and 119 came from the county (89 men and 30 women). A total of 61% of the men were under 45 years compared with only 27% of the women. There was very little difference between city and county with regard to the proportion of suicides under the age of 45 years.

The methods of suicide used in Cork were similar to those used in the rest of the country. Over 80% of the males used violent methods such as drowning, hanging and shooting and only 16% poisoned themselves. A total of 51% of the female suicides died by drowning and 33% by poisoning.

A total of 75% of the male suicides from a rural setting were single, 20% were married, 4% widowed and 1% separated. Of the women from a rural background, 40% were single, 33% were widowed, 3% were separated, and only 23% were married. Of male suicides in the city, 48% were single, 6% widowed, 6% separated and 36% married. Of female suicides in the city, 28% were single, 18% widowed, 10% separated and 42% married.

With regard to employment status, when the retired and those of uncertain status (e.g. rural farmers on the dole) were excluded, it was found that of the male suicides investigated in Cork, 58 were unemployed and 37 employed at the time of their deaths. Thirteen female suicides were unemployed compared with six employed. Thirty-one housewives were excluded from this calculation. There were also seven students (six men and one woman).

Discussion

The evidence presented here favours the view that the suicide rate in Ireland was genuinely low in the past but has significantly increased in the last 15 years. Three factors in particular support this.

Firstly, if in the past, deaths by suicide were incorrectly labelled as accidents or undetermined deaths, the official statistics would reveal this when compared year by year: as the pool of officially designated suicides increased, there would be a contraction in the number which received an open or an accidental verdict. However, while the suicide rate continued to show marked increases, the other verdicts did not show any definite trend until the 1980s when they showed a decrease of 24 per 1 000 000 population. Although this decrease may have contributed to the further sharp increases in suicide rates which occurred after 1980, it is not sufficient to account for the increase of 60 per 1 000 000 population in the suicide rate which occurred over the period of the study. Furthermore, the methods of suicide which showed the most remarkable increases were the violent ones of hanging, drowning and to a lesser extent shooting. CSO statistics (1970–85) show no evidence of an excess of these violent deaths attributed to causes other than suicide in the years before the rate started to rise.

Secondly, suicide no longer occurs mainly in the elderly. The suicide rate among Irish males under the age of 35 years increased by over 300% from 1970 to 1985. This appears to be part of an international trend. Increased suicide rates in men between the ages of 24 and 44 years have been reported from a number of countries (Sainsbury, 1986). McClure (1987) demonstrated that the rates for men in England and Wales increased by 37% in the 24–54-year age group between 1975 and 1984.

Thirdly, there has been a great increase in the suicide rates of the two largest cities. In the past, the urban suicide rate was always considerably lower than the rural rate in Ireland, and in the 1960s the suicide rates of Dublin and Cork were actually lower than the aggregate urban rates. From the mid-1970s, however, the suicide rate of these two cities, particularly Cork City, rose to a level well above the national average.

If it is accepted that the increase in the suicide rate is not due primarily to a change in recording practice then the causes must be sought elsewhere. A genuine rise in suicide could be determined by an increase in mental illness in the community. However, there is no evidence to support such a hypothesis in Ireland. In 1971, the number of first admissions to psychiatric hospitals was 8058 whereas in 1985 it was 8441, an increase of 5% when the population as a whole increased by 18% (Health Research Board, 1971–83). In our study only 34% of the male suicides and 43% of the female ones in Cork City were known to have had psychiatric treatment. The majority of these patients were well known to the services, having suffered from recurrent depression, chronic schizophrenia, alcoholism and severe personality disorder for a long time.

Information about previous psychiatric illness came from the files of the police and coroners and it may be an underestimate. The difference in regard to a history of psychiatric illness between urban and rural dwellers is marked and it is therefore possible that there may be unrecognised mental illness in rural areas. O'Hare & O'Connor (1987) suggest that where services are sparse, as in rural areas, it is the socially disruptive illnesses that attract treatment. Alcoholism, schizophrenia and personality disorders are disturbing to a community, whereas depression is more on a continuum with everyday life and therefore, when not disturbing anyone else, it may go unrecognised.

However, in the absence of any real evidence of an increase in psychiatric illness, the explanations of the increase in the suicide rate are likely to include changes in the cohesiveness in Irish society, in the confidence in a traditional value system and in occupational opportunities which provide a sense of self-worth and purpose. Social isolation probably also plays a role in the rural setting; 75% of the male suicides and 40% of the female suicides in Cork rural districts were single and a further 33% of the women were widowed.

Ireland is a less cohesive country now than it was 20 years ago. In support of this broad generalisation it has been shown that the illegitimacy rate, the number of indictable crimes and the number of admissions to hospital for alcohol-related problems more than doubled between 1970 and 1985. The marriage rate decreased during this period and, although there is no divorce in Ireland, the Labour Force Survey has shown that the numbers in the category 'married but separated' have increased each year after 1983, when they were first measured.

Durkheim (1897) was the first person in modern times to propose a link between a high suicide rate and a comparatively low level of integration in society. Sainsbury *et al* (1980) used a variety of social measurements to substantiate the link between the suicide rate and levels of social disorganisation. The present work confirms Sainsbury's findings in an Irish context. Although Ireland was materially poorer in the 1950s and 1960s, the suicide rate was lower at this time than in the 1970s and 1980s. Financial poverty itself, however, is unrelated to the rising suicide rates (Sainsbury, 1955); rather, it is the poverty of relationships and social opportunities that affect the rates. For many, particularly men, employment offers a ready-made set of relationships, providing the individual with a sense of purpose, obligation and reciprocal support outside the domestic unit. Throughout the past century the educational process has been geared to preparing the individual (man) for work in its various forms, inculcating in him, if possible, the idea that a worthwhile man is a man who works. In times of economic recession, when for cities such as Cork, the overall unemployment rate is 20% and in deprived areas considerably in excess of that, it is likely that unemployment becomes increasingly a cause for psychological distress. Indeed, Fogarty *et al* (1984) found that the unemployed in Ireland were a particularly demoralised and alienated group of people. Banks & Jackson (1982) found a strong association between unemployment and minor psychiatric disorder which disappeared when the individual found employment. Platt (1984) found there was significantly more unemployment, job instability, and occupational problems among suicides when compared with non-suicides. In our study in Cork, when we excluded housewives and those of uncertain status, over 60% of the suicides were unemployed. For the country as a whole, the relationship between the rising suicide rate and the rising unemployment rate is marked.

In conclusion, an increase in the official suicide rate in a country which is not due to a change in recording practice or an increase in the prevalence of psychiatric illness is likely to have a complex social aetiology. Aetiological factors probably include a decline in the cohesiveness of society as a whole and family life in particular, as well as a change in its value system regarding the consistency and conviction with which suicide is perceived as an amoral or an immoral act. The use of the educational system and the mass media to promote the idea of the benefits and obligations of the work ethic has negative effects for the individual particularly in times of recession when employment cannot be found. In such times many may accept their state with resignation, others complain, some may emigrate and a small although increasing minority may blame themselves, with fatal consequences. The change from institutional to community forms of psychiatric care cannot itself be blamed since the suicide rate had increased before this started, although for some years previously there had been a reluctance to admit people to long-stay beds. There is no doubt however that the mentally ill have, by the nature of their disabilities, been the least successful in accommodating to these rapid, multifarious social changes.

Acknowledgements

We are grateful to Coroners C T. O'Connell and C O'Riordan, Sgts Noel Dee and Morgan O'Flaherty, Mr Tony Perrott, Ms Eleanor O'Leary and to the staff of the Library at UCC and Cork Public Library.

References

BANKS, M. H. & JACKSON, P R (1982) Unemployment and risk of minor psychiatric disorder in young people – cross sectional and longitudinal evidence. *Psychological Medicine*, 12, 789–798.

—— (1978) The different incidence of suicide in Eire and in England & Wales. *British Journal of Psychiatry*, 132, 36–38.

CENTRAL STATISTICS OFFICE (1970–86*a*) Table 19. Deaths classified by cause (international detailed list, 9th revision 1975) and by sex and age *Report on Vital Statistics*. Dublin: CSO Stationary Office.

—— (1970–86*b*) Table XIV. Births per 1000 women in each age group in Ireland and England & Wales *Report on Vital Statistics*. Dublin: CSO Stationary Office.

—— (1970–86*c*) Table I Number of marriages per 1000 population in Ireland and neighbouring countries. *Report on Vital Statistics* Dublin: CSO Stationary Office.

—— (1971, 1981, 1986) *Census of the Republic of Ireland*. Dublin: CSO Stationary Office.

—— (1983–86) *Labour Force Survey*, Table C Dublin: CSO Stationary Office.

—— (1986) *Irish Statistical Bulletin*, Table VI, p. 209. Dublin: CSO Stationary Office.

Clarke-Finnegan, M. & Fahy, T. J. (1983) Suicide rates in Ireland. *Psychological Medicine*, 13, 385–391.

Cork Corporation (1984–86) *Cork Economic Monitor*. Cork: Town Planning Department, Cork Corporation.

Durkheim, E. (1897) *Le Suicide* (trans. (1952) by J. A Spaulding & C. Simpson as *Suicide: A study in sociology*). London: Routledge & Kegan Paul.

Eurostat (1982) *Review 1971–1980, Population and Social Conditions*, Section 3.4, p. 129. Luxembourg: Office for Official Publications of the European Communities

Fogarty, M., Ryan, L. & Lee, J. (1984) *Irish Values and Attitudes. The Irish Report of the European Value Systems Study*. Dublin: Dominican Publications.

Garda Siochana Stationary Office (1983–85). *The Report on Crime*. Dublin: Garda Siochana Stationary Office.

Health Education Bureau (1985) *Produktschap Voor Gedistilleerde Dranken Schiedam Netherlands* (reproduction). Dublin: Health Education Bureau.

Health Research Board (1971–85) *Activities of Irish Psychiatric Hospitals and Units*, Table 10. Dublin: Health Research Board.

Lester, D. (1972) Letter. *Medical Journal of Australia*, 941.

McCarthy, D. & Walsh, D. (1975) The under-reporting of suicide and the consequences for national statistics. *British Journal of Psychiatry*, 126, 301–308.

McClure, G. M. G. (1987) Suicide in England & Wales. *British Journal of Psychiatry*, 150, 309–314.

O'Hare, A. & O'Connor, A. (1987) Gender differences in treated mental illness in Ireland. In *Gender in Irish Society*. Galway: Galway University Press.

Platt, S. (1984) Unemployment and suicidal behaviour – a review of the literature. *Social Scientific Medicine*, 19, 93–115.

Sainsbury, P. (1955) *Suicide in London* (Maudsley Monograph no. 1). London: Chapman & Hall.

—— (1986) The epidemiology of suicide. In *Suicide* (ed. Alex A. Roy), p. 18. Baltimore, USA: Williams & Wilkins.

—— & Barraclough, B. M. (1968) Differences between suicide rates. *Nature*, 220, 1252.

——, Jenkins, J. S., Levey, A., *et al* (1978) *Suicide Trends in Europe: A Study of the Decline in Suicide in England & Wales and the Increase Elsewhere*. Geneva: WHO Regional Office for Europe.

——, —— & —— (1980) The social correlates of suicide in Europe. In *The Suicide Syndrome* (eds R. D. T. Farmer & S. R. Hirsch). London: Croom Helm.

Walsh, D. (1976) A century of suicide in Ireland. *Journal of the Irish Medical Association*, 69, 144–151.

*Michael J. Kelleher, MD, MPhil, BSc, MRCPsych, *Clinical Director, St Anne's Hospital & Clinic, Shanakiel, Cork, Ireland*; Maura Daly, MRCPsych, *Consultant Psychiatrist, St Anne's Hospital & Clinic, Shanakiel, Cork, Ireland*

*Correspondence

British Journal of Psychiatry (1990), 157, 539–550

Adult Affective Disorder and Early Environment

BRYAN RODGERS

Childhood precursors of symptoms of depression and anxiety were investigated in a national population sample of over 3000 men and women, aged 36 years. Early-life data had been collected prospectively for all subjects. A number of factors, differing for men and women, were found to be significant predictors of adult disorder. Some factors showed strong effects but tended to apply to relatively few individuals, while other more common circumstances had modest influences. Overall, early environment did not seem to hold great significance for adult affective disorder, although multiple disadvantages had a cumulative deleterious effect. There was little evidence of early benefits being protective.

The influence of childhood circumstances on adult mental health has been the subject of repeated empirical inquiry. Many authors (e.g. Brown *et al*, 1977; Tennant *et al*, 1980; Lloyd, 1980; Perris *et al*, 1986; Ragan & McGlashan, 1986) have drawn attention to the sustained interest in this topic, especially to the impact of the psychoanalytic tradition (Freud, 1913; Abraham, 1927; Bowlby, 1951), and the issue of early environmental effects on psychological development in general has an even longer history (Clarke & Clarke, 1976). More recent experimental research with animals, particularly the identification of sensitive periods in development (Newton & Levine, 1968; Rutter, 1981), has helped maintain this concern with early experience.

Investigations with human subjects have embraced a variety of independent variables and a range of adult outcomes, and have reached diverse conclusions. Regarding parental death during childhood and subsequent depression, for example, Langner & Michael (1963) reported that death of a parent before the age of seven years was associated with adult psychiatric symptoms but later deaths were not, whereas Brown (1961) found later childhood to be the more important. Other studies have implicated death of either parent (Dohrenwend & Figueiredo, 1983), father only (Hill & Price, 1967), or mother only (Munro & Griffiths, 1969), and childhood bereavement has been found to be more common in severely depressed patients (Beck *et al*, 1963) or invariant in relation to severity (Sethi, 1964). Some investigators have found no link between parental death and later disorder (Munro, 1966; Birtchnell, 1980; Roy, 1983; Faravelli *et al*, 1986; Hällström, 1987). Such inconsistencies thwart parsimonious explanation and the conclusions of three review papers published in the same year bear this out: "childhood loss of a parent by death generally increases depressive risk by a factor of about 2 or 3" (Lloyd, 1980); "parental death during childhood has not been established as a factor of etiologic significance in adult depression" (Crook & Eliot, 1980); "parental death in childhood has some effect, albeit weak, upon risk of adult depression . . . parental death in childhood on its own has little impact upon the risk of depressive illness in adult life" (Tennant *et al*, 1980).

There are three reasons why these results have special importance. Firstly, parental death is considered to be well defined and reliably reported (Granville-Grossman, 1968; Brown *et al*, 1977; Tennant *et al*, 1980). Secondly, it is rarely affected by the behaviour of children and, with the notable exception of suicide, cannot readily be attributed to a genetic predisposition to psychiatric disorder which later manifests in offspring (Paykel, 1982). Thirdly, the immediate emotional consequences of parental loss can be severe, albeit generally less prolonged than seen in the surviving parent (Marris, 1958). Failure to establish early bereavement as an aetiological factor in adult depression therefore has wider implications for the general hypothesis of long-term effects of adverse childhood experience.

Concern with the early environment is not confined to such major events as death of a family member or permanent separation from a parent. Disruption of parental attachment may be viewed as a gradation, with shorter separations and competing demands restricting the quantity of interaction with individual children. The argument can be extended further to encompass the quality of interaction between caretaker and child, and to include interactions with non-caretakers including siblings and peers.

Taking this broader perspective, three general processes can be identified whereby childhood experiences may influence adult mental health.

(a) Individual differences could develop in the emotional susceptibility to disorder. This is best indicated by morbidity in the absence of stress, but it would also be possible to see a disproportionate response to trivial provocation (Brown & Harris, 1978). (b) Early experiences could affect the likelihood and extent of adversity in adulthood. (c) Some individuals may become better able to cope than others when later adversity is encountered. These three mechanisms could operate concurrently and, furthermore, one particular childhood experience could initiate more than one process. They do, however, provide a crude framework for building hypotheses as to what early factors are expected to be influential. This paper is concerned with circumstances leading to symptoms of depression and anxiety, which frequently lead to contact with services, notably attendance at a general practice (Rodgers & Mann, 1986), and often impair functioning (Hurry & Sturt, 1981).

Early stress is a plausible candidate for affecting later emotionality. Such experiences as being admitted to hospital are implicated by the evidence of their immediate impact, which varies with age of occurrence (Prugh *et al*, 1953; Illingworth & Holt, 1955). Other stressful circumstances include inability to meet educational demands, changing schools, and poor relationships with peers. Home life can also be a major source of stress, whether due to general disharmony in the family, abuse, intolerance or overdemanding behaviour specifically directed at children, or to unreasonable constraints placed on their activities. Permanent loss of a parent also falls into this category but not exclusively, as it can also greatly harm a child's chances in life. For example, children from broken homes are at greater risk of their own marriage ending in divorce or separation, which may lead to further adversity such as financial hardship (Wadsworth & Maclean, 1986). Similarly, physical and intellectual disabilities can contribute to poor socio-economic prospects as well as being a source of stress during childhood (Pless, 1984).

The genesis of capacity to survive adversity, which involves such notions as self-esteem, locus of control and social support, seems less easy to identify. Social support, while emanating in part from an ability to form and maintain personal relationships and to solicit help when required, is particularly dependent upon the availability and willingness of others. Perhaps, too, the issue of nurturence of coping is clouded by the uncertainty and ambiguity surrounding the success of protective strategies and styles themselves; for example, confidence and independence may be beneficial but overconfidence and social isolation not. Nevertheless, there have been attempts to link patterns of upbringing with poor adult adjustment, even though mediating factors were not always explicitly identified (Munro, 1966; Parker, 1979, 1983).

Doubt as to the identity of protective attributes may be responsible for the tendency to fall back on broad demographic features as predictors of later mental health. The reviews by Granville-Grossman (1968) and Paykel (1982) demonstrate the interest in such features as parental age, family size, and birth order. Socio-economic circumstances have also been investigated (Power *et al*, 1986). This epidemiological approach can prove valuable if indicators of who is susceptible to adult disorder give insight into how this comes about.

A final area which may begin to arouse interest in relation to affective disorders is the role of pre-natal factors and birth complications. These have been linked with early behaviour and neurological status (Rogers *et al*, 1955; Knobloch *et al*, 1956) and to severe psychopathology with lasting consequences (Parnas *et al*, 1982), although their relevance to minor disorders is less certain (Drillien, 1964; Buka *et al*, 1988).

Several of these hypothesised early influences can be investigated in the Medical Research Council's National Survey of Health and Development (NSHD). This prospective longitudinal study has collected data on its subjects from birth to age 36 years. The approach has three particular methodological strong points: reports of early life are uncontaminated by knowledge of subsequent psychological development; healthy controls are readily available in the sample; and potential predictors of adult symptoms can be investigated concurrently using multivariate statistical techniques.

Method

The NSHD is a follow-up study of 5362 men and women born in one week of March 1946 in Great Britain. The original, larger birth sample consisted of all births from the week (Joint Committee, 1948); subsequent follow-ups were stratified and included all children of non-manual workers and agricultural workers and one in four of the remainder, with the exceptions of illegitimate and multiple births. Data collection has been attempted at frequent intervals for those still alive and resident in Great Britain; these cover home and family background, health, education, employment, behaviour, and attitudes. Further details of the data and the timing of contacts up to age 26 years have been given by Atkins *et al* (1981) and an overview of the study is presented by Wadsworth (1987).

The last completed follow-up was in 1982–83 (at age 36 years) and consisted of an interview and brief physical examination conducted by specially trained nurses. The interview included an assessment of psychiatric symptoms over the past month using a shortened version of the Present State Examination (PSE) of Wing *et al* (1974, 1977; Wing & Sturt, 1978). Interview procedures, the derivation of summary measures of psychiatric status, and their reliability and validity have been described by Rodgers & Mann (1986). Two indices are used here: the total PSE score and the index of definition (ID). The former is a sum of symptom ratings with a maximum of 68 and an actual distribution in the sample of 0–38. The ID is a scale of 1–8 which is conventionally dichotomised, with 'caseness' applying to IDs of 5–8 (Wing *et al*, 1978).

Although many studies have preferred 'caseness' to total PSE score as the outcome variable, the latter has proved more useful in the present study for the following reasons. (a) Appraisal of the validity of these measures showed that high total PSE score (10 or more) was as good a predictor of recent treatment seeking and of subjective assessment of chronic illness as was an ID of 5 or more (Rodgers & Mann, 1986). (b) There was no indication of discontinuity in the scale of total PSE score, either when predicting these same outcomes (Rodgers & Mann, 1986) or when predicted by measures of temperament from earlier life (Rodgers, 1990*a*). (c) When presenting findings for small subgroups of the sample, mean total PSE score was a more robust summary statistic than rate of caseness (Rodgers, 1990*a*). Points (a) and (b) come as no surprise given that studies investigating changes in PSE ratings over time show that high subthreshold scores are predictive of subsequent caseness (Brown *et al*, 1986). In general these considerations underline the view that "the decision as to where sub-clinical disturbance ends and being a psychiatric case begins is, in the last analysis, arbitrary" (Goldberg & Huxley, 1980, p. 3).

Variables in six areas from the earlier years of the study were considered to be of potential influence on adult mental health.

Perinatal factors. Little was known about pre-natal factors and birth complications but birthweight would in part reflect some pre-natal influences. Parents' ages at birth were also known: clearly they could have a lasting influence on the environment throughout childhood and are particularly linked with parity (Granville-Grossman, 1968). Duration of breast-feeding was investigated because it has shown an association with intellectual development (Rodgers, 1978).

Socio-economic circumstances. These were indicated by parents' educational qualifications, father's social class, housing tenure, and an index of material home conditions as rated by health visitors (Rodgers, 1978). Information from visits at four years old was used in the analyses presented here but similar findings were obtained for other ages.

Family structure and disruption. Family size was calculated from the number of older siblings (at the time of birth) and the number of subsequent siblings born up until the survey members' 15th birthday. Slater's (1962) birth-order index was grouped into categories of only children, other first-born, last-born, and intermediate. The interval between the subject's birth and the birth of next sibling was also known. The date of divorce or separation of parents was obtained from reports near to the occasion, as was the occurrence of parental and sibling deaths, when most reports were confirmed by official records. Age at the time of bereavement was grouped into bands corresponding to stages of schooling, following the discussion of Dohrenwend & Figueiredo (1983): pre-school (before age 5), primary school (5–11 years), secondary school (12–17 years), and post-school (18–26 years). Shorter-term separations from parents were identified in several ways. Questions about the father's absence from home due to his employment were asked regularly during childhood. The time of a return to work by the mother during the pre-school years was also established. Inquiries were made about longer separation from the mother but only up to age six.

Childhood illness. Illness and injury were known from parental interviews, school-attendance records, periodic medical examinations, and hospital in-patient records. Serious illness at ages 0–5, 6–10 and 11–15 was identified from these sources (Wadsworth, 1986). The length of any hospital admission, the age of the subject at admission and the diagnosis made were all recorded.

Parental behaviour, attitudes and health. At age four, health visitors rated each mother's 'management and understanding' of the child as average, among the best, or among the worst. (They considered the mother's sympathy and understanding rather than the cleanliness of the child or home.) An index of parental interest in the child's education was derived from interviews with mothers and from information from primary schools (Douglas, 1964). A brief retrospective account of family faith or religious denomination was obtained at a 26-year interview (Wadsworth & Freeman, 1983). Information on parental health came from mothers' reports of their own and their husbands' complaints (when the survey child was 15 years) which included 'nerves'. At this time the mother also completed the six neuroticism items of the short Maudsley Personality Inventory (MPI; Eysenck, 1958).

Schooling. Educational progress was assessed by standardised attainment tests at ages 8, 11 and 15 years (Pidgeon, 1964, 1968). Receipt of special educational help including placement in a special class or school was known for the primary and secondary years. Data on provision also included the number of primary and secondary schools attended and the type of secondary schools attended.

When the above factors were considered as predictors of adult total PSE scores, linear-regression analysis and analysis of variance were utilised (using STEPWISE and GLM procedures of SAS). Parallel analyses with a logarithmic transformation of PSE score (which is positively skewed) were also carried out but made little difference to the findings. Caseness (ID = 5 +) was also used as an outcome in bivariate analysis when χ^2 tests (or the Fisher exact test if appropriate) were applied.

Results

Successful PSE interviews were obtained from 3293 survey members contacted in 1982–83 (Rodgers & Mann, 1986). This figure represented 75.7% of those assumed to be still living in Britain (i.e. excluding death and known emigration). The true response rate would have been higher as not all emigrations were known to the study. Bias as a result of selective drop-out was known to be slight for the first 26 years of the study (Atkins *et al*, 1981) but there were significant losses of former pupils of educationally subnormal schools (Rodgers, 1979) and others with very poor literacy skills (Rodgers, 1986). More recently there was a low contact rate for those known to have had a serious psychiatric illness, particularly schizophrenia (Rodgers, 1990*a*). Response rates for those of high measured neuroticism, the best single predictor of adult PSE scores, were typical of the general sample (Table I).

TABLE I
Contact rate at 36 years by neuroticism score at age 16

	Neuroticism score				
	0–3	*4–6*	*7–9*	*10–12*	*Total*
Contact rate	71.5%	71.8%	74.4%	72.1%	72.2%[1]
No. of subjects	1014	1157	733	934	3838[1]

1. Of those contacted at 36 years, 522 had not completed the neuroticism scale.

Of 1640 men interviewed, 62 (3.8%) were rated at caseness levels as compared with 142 of the 1653 women (8.6%). These figures were unchanged when corrected for the sample stratification (according to work status of parents). In terms of total PSE scores, men had a mean of 1.80 and women 3.04 before correction and 1.83 and 3.09 respectively after adjustment. As well as this familiar sex difference, statistical interactions were found between early factors and sex. Results for men and women were therefore analysed separately.

Perinatal factors

There was no discernible trend of symptoms in relation to birthweight for either sex. Those with very low birthweights (under 5 lb, 2.3 kg) even had slightly fewer symptoms on average than their peers but not significantly so. There would be more low-birthweight survivors in later-born cohorts, which could produce different findings.

Sons born of the oldest fathers (44 years or more) had higher mean PSE scores than those with younger fathers (2.65 v. 1.67, $P=0.007$), a difference corresponding to a doubling in the rate of caseness. Age of mother at birth was also significant for men but not when father's age was taken into account. In women there was a similar pattern of more symptoms for those with older fathers but this was not statistically significant, and age of mother showed no association with PSE assessment.

Duration of breast-feeding in infancy showed no link with adult mental health.

Socio-economic circumstances

Social class of father was unrelated to subsequent symptoms for men, and for women the only significant finding was of higher mean total PSE scores in those from social class V (4.06 v. 2.95 for other social classes, $P=0.02$).

Men who had lived in council housing (at age four years) had a mean PSE score of 2.09, whereas others had a mean score of 1.69 ($P=0.04$); for women the figures were 3.48 and 2.86 ($P=0.02$), respectively. Similar differences were found in the rates of caseness, being 5.5% and 3.3% respectively for men ($\chi^2=3.98$, $P=0.05$), and 11.7% and 7.4% for women ($\chi^2=7.60$, $P=0.006$). Somewhat surprisingly, given these findings, the overall index of material home conditions showed no similar associations, but overcrowding was important. Mean total PSE score for women brought up in households with over two people per room was 4.18 versus 2.95 for others ($P=0.009$). The difference was less for men and of only borderline significance.

Although low social class, tenure and crowding were inter-related, all made an independent contribution when considered together in an analysis of women's symptoms.

The educational qualifications of parents did not show any link with later mental health of their children. It may be significant that over 60% of parents had no qualifications and received elementary education only, allowing no discrimination at one end of the spectrum.

Family structure and disruption

Effects of family size and birth order were analysed in a number of ways. When complete family size and birth rank were investigated together it was family size which appeared the important factor, larger families being associated with greater frequencies of symptoms. For men the difference was small, but for women the PSE scores of those with more than one sibling (mean = 3.31) were higher than those from smaller families (mean = 2.73, $P=0.02$). Birth order was also found to be significantly associated with women's symptoms. The four categories of birth order (only children, first-born, last-born and intermediate) are strongly linked with family size, however, and in practice it made little difference which of these indices was utilised. Finally, the interval between the subject's birth and the arrival of the next sibling was unrelated to subsequent mental health. Family size was associated with social class, housing tenure and, of course, with overcrowding. When these four variables were used in combination to predict women's PSE scores, the first failed to make a significant independent contribution.

There was no evidence that divorce or separation of parents resulted in later affective disorder for their sons. This was so at whatever age the break occurred. For daughters, relatively high frequencies of symptoms were found for breaks occurring at all ages through to early adulthood (Table II). The highest rate of caseness was found for those who experienced breaks before age five, but it should be noted that prevalence is based on small numbers. Clearly, several mechanisms could be involved in the links between family breakdown and development of these children but one factor which did not appear important in this respect was socio-economic circumstances of families, either before the break or after. Also, where children

TABLE II
Mean total PSE scores and rate of 'caseness'

	No. of subjects	*Mean PSE score*	*Rate of 'caseness'*
Parents separated when daughter aged:			
<5 years	52	4.54	26.9%
5–11 years	32	3.22	12.5%
12–17 years	23	4.43	17.4%
18–26 years	20	4.95	15.0%
Parents not separated	1526	2.94	7.7%
Father died when son aged:			
<5 years	21	1.67	4.8%
5–11 years	34	3.76	8.8%
12–17 years	47	2.45	2.1%
18–27 years	113	1.87	4.4%
Father not dead	1424	1.73	3.7%
Temporary separation of sons from mothers before 6 years of age:			
Not separated for 1 month or more	1352	1.63	3.3%
Separated for 1 month or more	151	2.91	7.9%

stayed with their mother (as usually happened) the introduction of a step-father did not appear to have any effect. The number of children who became separated from their mother was small and they were heterogeneous as regards subsequent caretakers, but their adult status was no worse than that of those staying with their mothers. However, there was a high drop-out from the study of children who were permanently separated from their mothers, particularly those who received some form of institutional or foster-care, and this negative finding should be viewed with caution.

The death of a sibling did not appear to have any long-term consequence for mental health. Overall the loss of a parent by death before the age of 18 did not have a significant adverse effect on women, but the 139 men who had experienced such a loss had raised mean PSE scores (2.52 v. 1.73, $P = 0.008$). This difference was seen predominantly in those who had lost their father (which was more than twice as likely as mother's death) and was particularly marked in the group experiencing the loss when aged 5–11 years (Table II). The 'effect' of father's death was not seen in the boys whose mothers remarried. The smaller increase in scores of men who had lost their mother was restricted to losses during later childhood but this failed to reach statistical significance. Although findings for women were negative this should not be interpreted as indicating a definite sex difference. Similar trends were seen in women: an association with loss of the parent of the same sex occurring at ages 5–11 years and an association with death of the parent of the opposite sex at later ages (12–17 years), with no apparent consequence of a loss during the pre-school years.

Clearly, older fathers had a greater likelihood of dying during the subject's childhood years, but father's age and death of father had independent effects when considered together.

Overnight absence of the father caused by his employment appeared to have no long-term influence on the PSE scores of sons or daughters. Similarly, daytime absence of mothers who returned to work before the survey child started school was not a significant factor. Longer separations from the mother (for all reasons) up until age six seemed to have no adverse consequences unless they lasted more than four weeks; these were often caused by parental illness or the child entering hospital (Table II). The 151 boys so affected had mean PSE scores of 2.91 at age 36 compared with 1.63 for their peers ($P < 0.001$). Figures for girls were less striking: 3.76 ($n = 147$) compared with 2.98 ($P = 0.05$), respectively. Boys placed in residential care or with foster-parents when a temporary break occurred had particularly high PSE scores as adults (mean = 4.50; $n = 22$; $P < 0.001$). Four of these 22 met PSE criteria for caseness. This was five times the rate in the total sample and is statistically significant even with such small numbers ($P = 0.008$, Fisher exact test).

Childhood illness

Childhood illness was associated with adult symptoms but only under certain circumstances. For men, significantly higher PSE scores were found following admission to hospital in early life if the stay was longer than four weeks. This group was a subgroup of those who had been temporarily separated from their mothers and did not differ from those separated for other reasons. Women with a serious illness at any stage of childhood had raised PSE scores. This was attributable to a small group ($n = 22$) with a history of chronic illness or disability who had mean scores of 6.23 compared with 3.00 for their more fortunate peers ($P < 0.001$). (A similar trend was found for men but was not statistically significant.) Disabilities included congenital abnormalities of the limbs and heart, spina bifida, polio and severe asthma, conditions which would be expected to persist into adulthood. When they were excluded from analysis the only indication of further influence for women was in connection with hospital admissions during the teenage years, but there was no systematic trend with either the number of admissions or the length of stay. The 300 girls admitted between 12 and 17 years had a mean PSE score of 3.63 at age 36 compared with 2.91 for 1337 who were not admitted ($P = 0.01$) (data missing for 16 subjects). Omission of the chronically sick group of women was found to reduce the previously described 'effect' of temporary separations from mothers to a non-significant level.

School absenteeism was associated with adult symptoms for women but the relationship only held for the later years of schooling. Absenteeism was intended as a proxy for physical morbidity, but towards the final years of schooling it turned out to show highly significant relationships with measures of personality and behaviour in school, characteristics known to be predictive of adult psychiatric status (Tyrer & Tyrer, 1974; Robins & Ratcliff, 1980; Rodgers, 1990*a*).

Parental behaviour, attitudes and health

Little difference was found between the mean PSE scores of those brought up by mothers who were rated among the best in management and understanding of their child and those whose mothers were considered average in this respect, whereas children of the worst mothers had very high scores. The number of individuals concerned was small, however. Three out of 15 such boys reached caseness level on the 36-year PSE, as did 5 out of 16 girls, their mean PSE scores being 3.1 and 6.3 respectively. The latter was significantly different from the scores of other women ($P = 0.004$).

Parental interest in education was only associated with adult outcome for women and was only influential insomuch as the children of those in the bottom decile (i.e. least interested parents) had high PSE scores at age 36 (mean 3.84 v. 2.93 for other women, $P = 0.02$).

Type of religious faith of upbringing was unrelated to adult symptoms for both sexes, but women who said they had no faith of upbringing had more symptoms as adults (mean = 3.85, $n = 164$, $P = 0.01$). As just over 10% fell into this category it was clear that a fairly liberal view had been taken of what constituted a religious upbringing.

A number of aspects of parents' health were predictive of their children's symptoms. Fathers' ill-health, although having no long-term consequences for sons, was associated with high total PSE scores for women. The daughters of men with physical complaints ($n = 367$) had mean scores of 3.67 versus 2.74 for others ($P < 0.001$), and those whose fathers had mental-health problems ($n = 54$) had higher scores still (mean = 4.75, $P = 0.002$). Mothers' self-reports of nervous complaints, but not physical illness, were predictive of sons' PSE scores (2.40 v. 1.66, $P = 0.01$), and both their physical and mental problems were associated with high scores for their daughters (means of 3.42 ($n = 495$) and 3.72 ($n = 164$) respectively, $P = 0.007$ and $P = 0.03$). Not surprisingly, mothers' neuroticism scores were also associated with subjects' later PSE scores. Sons of the least stable mothers had scores of 2.74 at age 36 years ($n = 103$) compared with 1.74 for their peers ($P = 0.002$), and for daughters the corresponding values were 3.98 ($n = 90$) and 2.93 ($P = 0.03$). There was considerable overlap between self-reports of nervous complaints (and to a lesser extent physical illness) and high scores on the neuroticism scale for mothers, and the latter was found to be the better predictor.

Schooling

There was a clear sex difference in the relationships between attainment test scores from the school years and PSE symptoms at age 36. No significant associations were found for men whereas most tests were useful predictors for women. The test scores were not discriminating over their entire range but only towards their lower end, the optimum cut-off being around 80 points (mean = 100, s.d. = 15). Intercorrelations between scores were high and the relationship found with general ability at age 15 (an aggregate of verbal and non-verbal intelligence) subsumed the associations with other tests. The 102 women with 80 points or less on the general-ability scale at age 15 had mean adult PSE scores of 4.57, significantly more than the 2.89 found for other women ($P < 0.001$).

Type of school appeared an unimportant factor except when pupils had attended a special school. Boys who received special educational help ($n = 55$) were subsequently found to have mean PSE scores of 2.80 compared with 1.76 for others ($P = 0.02$) and the corresponding figures for girls were 6.02 ($n = 47$) and 2.95 ($P < 0.001$). It was striking that 15 of these 47 girls met the criteria for caseness at age 36, including four out of eight girls who had been classified as educationally subnormal. There was substantial overlap between the group with low ability scores and those receiving special help, but the latter factor was the more important as an indicator of adult psychiatric status. This could reflect a tendency to exclude children with psychosocial problems from mainstream educational provision or it may be that placement in special classes and schools itself has an adverse effect, either contemporaneously or brought about subsequently by poorer chances in life. The explanation that these pupils have poor outcomes in adulthood because they are from poor socio-economic backgrounds and are subject to a high incidence of family disruption (Stein & Susser, 1960) was partially supported by the finding that these factors accounted for some of the disadvantage for boys receiving special help, but they did not account for the poor outcome for girls.

One final environmental influence included in this investigation was that of changing schools. This seemed unimportant during secondary school but, for girls only, it appeared that those who attended more than one primary school had higher symptom scores at age 36 years (mean = 3.32, $n = 633$) than their peers who were at the same school throughout (mean = 2.86, $n = 885$). The difference was small and just significant ($P = 0.05$), but even the modest adjustment for an association with parental divorce/separation (girls from such families being more likely to change schools) attenuated this relationship.

TABLE III

Early environmental factors associated with total PSE score at age 36

	No. of subjects	*Unadjusted deviation*	*Adjusted deviation*	F *to omit*	P
Men (n = *1640*)[1]					
Father's age at birth					
up to 42 years	1363	−0.13	−0.09	5.44	0.020[2]
over 42 years	89	0.85	0.72		
missing data	188	0.50	0.34		
Death of father					
5–11 years old[3]	30	2.23	2.04	11.43	<0.001
others	1610	−0.04	−0.04		
Temporary separations					
(i) over 4 weeks	151	1.11	0.87	11.92	<0.001[2]
others	1352	−0.17	−0.13		
missing data	137	0.48	0.36		
(ii) in care/fostered	22	2.70	2.01	7.87	0.005
others	1618	−0.04	−0.03		
Mother's neuroticism					
least stable	103	0.94	0.94	8.82	0.003
others	1537	−0.06	−0.06		
Schooling					
special school or class	55	1.00	0.77	3.05	0.081
no special schooling	1585	−0.03	−0.03		
Women (n = *1342*)[4]					
Parents' marriage					
divorced/separated	94	1.80	1.61	13.31	<0.001
intact	1248	−0.14	−0.12		
Health 0–15 years					
serious chronic illness	20	3.64	3.08	9.92	0.002
others	1322	−0.06	−0.05		
Mother's understanding					
among the worst	15	3.64	2.82	6.17	0.013
average or best	1327	−0.04	−0.03		
Mother's neuroticism					
continuous (0–6)	—	×0.29	×0.23	9.08	0.003
Father's mental health					
'nerves' reported	56	1.94	1.47	6.38	0.012
others	1286	−0.08	−0.06		
Father's physical health					
illness reported	339	0.81	0.67	10.20	0.001
others	1003	−0.28	−0.23		
Religious upbringing					
no faith reported	147	1.14	0.83	5.76	0.017
faith reported	1195	−0.14	−0.10		
Schooling					
special school or class	38	2.70	2.25	10.15	0.002
no special schooling	1304	−0.08	−0.07		

1. Total $R^2 = 0.038$.
2. Excluding cases with missing data.
3. Omitting those with step-fathers.
4. Total $R^2 = 0.063$; data missing for 311 women.

Combined influence of early environmental factors

The factors found by the previous analyses to be useful indicators of disorder in adulthood were considered in combination in order to establish which were the best predictors and to assess the strength of overall prediction. All variables from the six general areas which had been found to make an independent contribution in respect of total PSE score were used for this purpose, but those which failed to have a significant 'effect' in this multivariate analysis were excluded from the final models.

Men

Three variables were omitted from the model: housing tenure, overcrowding in early life, and mother's nervous complaints during the secondary-school years. The loss of the socio-economic factors from the analysis does not imply that such circumstances are unimportant but suggest that it is the other, more specific adverse characteristics of childhood which are responsible for the higher rates of symptoms found in those from poorer backgrounds. As already mentioned, self-reports of nervous problems by mothers were largely overshadowed by the measurement of neuroticism from the MPI.

The remaining six variables found to have a significant 'effect' are shown in Table III, along with the deviation values for each category. A category for missing data was included for two variables because of the atypical nature of those groups in respect of both the dependent variable and other independent variables. The statistical significance of these variables (temporary separations from the mother and father's age) was estimated following the exclusion of the missing cases, however. Special schooling was retained in the model because it also made a significant independent contribution when these missing cases were omitted. The small number of survey members who had received such provision meant that the estimation of its association with later symptoms was influenced by quite small sample fluctuations.

Interaction terms were non-significant but it was not assumed that this necessarily implied additivity of the main effects, particularly as relatively few individuals encountered combinations of adverse circumstances. Such combinations were the focus of a supplementary analysis which showed that the boys so affected did indeed have high symptom scores as adults which were consistent with additivity of the separate factors. An additional interaction was examined even though one component of this was non-significant as a main effect – Roff (1974) has suggested that concurrent mental health problems in mother and father are particularly deleterious, but no support for this was found.

TABLE IV
Rate of 'caseness' by level of childhood adversity

	Level of adversity[1]			
	Low <			> *High*
Men (n = *1640*)				
Rate of 'caseness'	3.0%	4.7%	11.5%	16.7%
No. of subjects	1281	277	52	30
Women (n = *1342*)[2]				
Rate of 'caseness'	4.2%	7.8%	12.8%	32.3%
No. of subjects	473	625	179	65

1. Calculated from the regression coefficients of factors remaining in the final model (see Table III) Arbitrary cut-off scores were used to indicate low–high levels.
2. Data missing for 311 women.

The measure of variance explained gives a rather intangible impression of the overall strength of prediction and the form of the analysis does not indicate whether the associations found can be taken to apply to prediction of extreme symptom levels. To look at this further, weightings of the early environmental factors (derived from the multivariate linear model) were summed for individuals and rates of caseness were calculated for several levels of early adversity, as shown in Table IV. The highest level of adversity necessarily involved combinations of the six factors considered. The rate of caseness in this group, 16.7%, was over five times that found for those who experienced little adversity in early life. By taking an arbitrary cut-off point on the scale of adversity it was possible to calculate a measure of association in the form of attributable risk per cent (MacMahon & Pugh, 1970). With the division between the top two and bottom two groups this gave a figure of 13.4%.

Women

A similar analysis for women was carried out, and again it was found that variables reflecting poor socio-economic circumstances dropped out of the multivariate model. Other factors of borderline significance were also lost from the final model, including hospital admissions during the teenage years, parental interest in education, and number of primary schools attended. Again mother's health no longer made an independent contribution when other factors, including her own neuroticism score, were considered concurrently. The neuroticism score was found to show the strongest association with daughters' symptoms when it was used as a continuous variable. Table III shows the 'effects' of the eight factors which were significant in the final model. In total they explained 6.3% of the variance in total PSE scores. Again there were no significant interaction terms but supplementary analysis indicated that those with experience of multiple adversity had rather more symptoms in adulthood than anticipated from the separate effects of these factors. For example, where both parents had been reported as having mental-health problems very high PSE scores were observed (mean = 6.1), but only 19 women fell into this category. Rate of caseness by levels of early adversity is shown in Table IV. The rate in the most disadvantaged group, 32.3%, was over seven times that in the least disadvantaged group, and the index of attributable risk per cent for the two highest levels of adversity combined was 25.4%.

Discussion

There are a number of aspects of the design of this study which need to be considered. That it used a particular cohort as its sample may limit the extent to which results can be generalised. It may be, for example, that the survival of low-birthweight babies in subsequent cohorts could bring about different relationships between perinatal factors and adult mental health. The significance of parental death may also have varied between generations: not only has it generally declined, but also some cohorts have been affected more than others by periods of war. The opposite trend of a substantial increase in the divorce rate may have modified the impact of this event, too. If equivalent findings could be obtained from future follow-ups of the National Child Development Study (1958 British-birth cohort) they would yield useful comparisons.

It must also be acknowledged that the data collected during the early years of the study were not chosen for their potential in predicting symptoms in adulthood, and stronger associations could no doubt be obtained using different independent variables. The lack of information on parenting behaviour was most notable. However, if interest lies in the possibility of screening higher-risk children as a preventive measure then it is unlikely that more detailed information would be available.

The strength of prediction would also have been influenced by the nature of the dependent measures (total PSE score and ID), primarily because of the focus of attention on the month before interview. Even though many individuals rated highly on the PSE considered themselves as having long-term complaints (Rodgers & Mann, 1986), the liability of neurotic symptoms in a population study (Henderson *et al*, 1981) is such that sampling a short time span necessarily limits correlations with temporally distant factors. The associations reported in this paper are therefore likely to understate the relationship with chronic disorder.

Finally, the reported associations do not necessarily describe causal processes and, even when this is likely, the complete chain of influence is unlikely to be elucidated. It is not clear, for example, whether early separations from parents have an immediate and lasting impact on the individual's psyche or whether it is the disruption of interactions and relationships accompanying such events that is important (Rutter, 1981; Paykel, 1982). In either case the effect could be mediated by bringing about further adversity in adulthood or by establishing a personal vulnerability to adversity.

When looking at the early factors implicated by these analyses it was the areas of family disruption and parental attitudes and behaviour which achieved prominence. However, separation from parents *per se* did not appear to be an important factor, as attested by the weak associations between parental death and later disorder. That parental divorce or separation should be associated with neurosis in women and, as demonstrated previously (Wadsworth, 1979), with delinquency in young men suggests that it is circumstances surrounding the event rather than the break itself which are significant (Harris *et al*, 1986; Bifulco *et al*, 1987; Breier *et al*, 1988). A similar interpretation could be applied to temporary separations, which were often accompanied by illness of either the child or a parent (usually the mother) or were precipitated by other difficulties in the family. Although admission to hospital in childhood has been particularly linked with short-term and medium-term disturbances of behaviour (Douglas, 1975; Quinton & Rutter, 1976), the suggestion here was that its long-term consequences were similar to those of other temporary separations.

While the correlation between total PSE scores and mothers' neuroticism scores and their association with fathers' nervous complaints were consistent with a degree of genetic inheritance, it could also be that parental behaviour has a lasting impact on offspring or that parents' psychological status reflects stressful circumstances within families which are the origins of later disorder in the children. This last interpretation draws support from the finding that high rates of persistent behavioural disorders in the children of psychiatric patients have been found particularly in those families where hostility and discord were evident (Rutter & Quinton, 1984). It would be of interest to know whether such family disturbance is also implicated in the poor outcome for children of very low academic achievement. The behavioural and temperamental features known to accompany learning difficulties in children (Rutter & Graham, 1970) could be precursors of adult disorder. Alternatively, problems arising in adulthood as a consequence of poor basic skills or even the attempt to conceal disability (Saunders & Barker, 1972) may place stress on individuals. Although there have been many follow-up studies of pupils from special schools (Tizard, 1974), psychiatric disorders have rarely been considered in these investigations; the high risk of affective disorder could make this a profitable area of research.

A comment on the different environmental factors implicated for men and women seems appropriate, but a comprehensive explanation of the findings is hard to formulate. Such results could arise because boys and girls are susceptible to different influences, perhaps because of differences in constitution (Rutter, 1983). It is also possible that the immediate psychological consequences of early experiences are similar for the sexes but that these lead to different adult outcomes. This second interpretation is consistent with the sex differences found in this study in the prediction of adult disorder from childhood behaviour and temperament (Rodgers, 1990*a*). A third explanation is that the early environment has implications for future life circumstances and adversity, and that risk and protective factors in adulthood differ for men and women. This will be a topic for further study. Whatever the mechanisms involved, it is notable that the strength of association with adult disorder was greater for women than men, which is the reverse of that usually reported for shorter-term influences on childhood behaviour (Rutter, 1983).

Taking an overall view of these results it seems that even an allowance for methodological shortcomings (which tend to understate relationships) does not lead to the conclusion that early environment holds great significance for adult affective disorder, although it was the case that high rates of disorder were found in those who experienced multiple disadvantages in childhood. When individual factors did appear to have strong associations with adult symptoms this was for rather small groups with particularly adverse circumstances – those with mothers described as the poorest carers or children placed in residential care or in special schools, for example – but the majority of adults ascribed caseness levels on the PSE were not from these groups. The relationships observed were reminiscent of aspects of the vitamin-model approach to mental health described, in another context, by Warr (1987). The tendency for those at the extremity of a continuum of childhood disadvantage to show poor adult mental health stands in marked contrast to the steadily graded associations of early environment with intellectual development (Douglas, 1964; Rodgers, 1978) or with physical growth (Kuh & Wadsworth, 1989). Although there appear to be circumstances which are best avoided to minimise the risk of later neurosis there is little to suggest that positive efforts can be made to nurture protection from adult disorder. Perhaps more sensitive measures of the childhood environment can be used to challenge this conclusion and it is hoped that additional data will allow a fuller investigation.

There are, in addition, some positive implications of these findings. The described associations deserve more detailed exploration to elucidate the pathways involved. It can be asked, for example, whether the early factors identified have any immediate effects on childhood behaviour and temperament, and there is some evidence of this (Rodgers, 1990*a*). It is also possible to examine whether early environment and recent factors interact to bring about adult neurosis in a manner which is consistent with notions of vulnerability (Brown & Harris, 1978; Rodgers, 1990*b*). It will be important, too, to investigate continuities between adversity in childhood and risk factors in adulthood (Rutter, 1984) which could contribute to the links between early environment and adult disorder. There is a likelihood that several mechanisms are involved in bringing about the observed associations.

References

Abraham, K. (1927) Notes on the psycho-analytical investigation and treatment of manic–depressive insanity and allied conditions In *Selected Papers on Psycho-analysis*. London: Hogarth Press.

Atkins, E., Cherry, N., Douglas, J. W B., *et al* (1981) The 1946 British birth cohort: an account of the origins, progress, and results of the National Survey of Health and Development. In *Prospective Longitudinal Research: an Empirical Basis for the Primary Prevention of Psychosocial Disorders* (eds S. A. Mednick & A. E Baert). London· Oxford University Press

Beck, A. T., Sethi, B. B. & Tuthill, R. W. (1963) Childhood bereavement and adult depression *Archives of General Psychiatry*, **9**, 295–302.

Bifulco, A. T., Brown, G. W. & Harris, T O. (1987) Childhood loss of parent, lack of adequate parental care and adult depression: a replication. *Journal of Affective Disorders*, **12**, 115–128.

Birtchnell, J. (1980) Women whose mothers died in childhood: an outcome study. *Psychological Medicine*, **10**, 699–713.

Bowlby, J. (1951) *Maternal Care and Mental Health* Geneva: World Health Organization.

Breier, A., Kelsoe, J. R., Kirwin, P. D., *et al* (1988) Early parental loss and development of adult psychopathology *Archives of General Psychiatry*, **45**, 987–993.

Brown, F (1961) Depression and childhood bereavement. *Journal of Mental Science*, **107**, 754–777.

Brown, G. W., Harris, T. & Copeland, J R (1977) Depression and loss. *British Journal of Psychiatry*, **130**, 1–18.

—— & —— (1978) *Social Origins of Depression: A Study of Psychiatric Disorder in Women.* London: Tavistock.

——, Bifulco, A., Harris, T , *et al* (1986) Life stress, chronic subclinical symptoms and vulnerability to clinical depression. *Journal of Affective Disorders*, **11**, 1–19.

Buka, S. L., Lipsitt, L P & Tsuang, M T (1988) Birth complications and psychological deviancy: a twenty-five year prospective inquiry. *Acta Pediatrica Japonica Overseas Edition*, **30**, 537–546.

Clarke, A. D. B. & Clarke, A. M. (1976) The formative years? In *Early Experience: Myth and Evidence* (eds. A. M. Clarke and A. D. B. Clarke) London: Open Books.

Crook, T. & Eliot, J. (1980) Parental death during childhood and adult depression: a critical review of the literature. *Psychological Bulletin*, **87**, 252–259

Dohrenwend, B. P & Figueiredo, J M. (1983) Remote and recent life events and psychopathology. In *Origins of Psychopathology. Problems in Research and Public Policy* (eds D. F. Ricks & B. S. Dohrenwend). New York: Cambridge University Press.

Douglas, J. W B. (1964) *The Home and the School.* London: MacGibbon and Kee.

Douglas, J. W. B. (1975) Early hospital admissions and later disturbances of behaviour and learning. *Developmental Medicine and Child Neurology*, **17**, 456–480.

Drillien, C. M. (1964) *The Growth and Development of the Prematurely Born Infant*. Edinburgh: E. & S. Livingstone.

Eysenck, H. J. (1958) A short questionnaire for the measurement of two dimensions of personality. *Journal of Applied Psychology*, **43**, 14–17.

Faravelli, C., Sacchetti, E., Amboretti, A., *et al* (1986) Early life events and affective disorder revisited. *British Journal of Psychiatry*, **148**, 288–295.

Freud, S. (1913) The claims of psycho-analysis to scientific interest: the interest of psycho-analysis from a developmental point of view. In *The Complete Psychological Works of Sigmund Freud*, vol. 13 (ed. J. Strachey, 1955). London: Hogarth Press.

Goldberg, D. P. & Huxley, P. J. (1980) *Mental Illness in the Community*. London: Tavistock.

Granville-Grossman, K. L. (1968) The early environment in affective disorder In *Recent Developments in Affective Disorders* (eds A. Coppen & A. Walk). London: Royal Medico-Psychological Association.

Hallström, T. (1987) The relationships of childhood socio-demographic factors and early parental loss to major depression in adult life. *Acta Psychiatrica Scandinavica*, **75**, 212–216.

Harris, T , Brown, G. W. & Bifulco, A. (1986) Loss of parent in childhood and adult psychiatric disorder: the role of lack of adequate parental care. *Psychological Medicine*, **16**, 641–659.

Henderson, S., Byrne, D. G. & Duncan-Jones, P. (1981) *Neurosis and the Social Environment*. Sydney: Academic Press.

Hill, O. W. & Price, J. S. (1967) Childhood bereavement and adult depression. *British Journal of Psychiatry*, **113**, 743–751.

Hurry, J. & Sturt, E. (1981) Social performance in a population sample: relation to psychiatric symptoms. In *What is a Case?* (eds J. K. Wing, P. Bebbington & L. N. Robins). London: Grant McIntyre.

Illingworth, R. S. & Holt, K. S. (1955) Children in hospital: some observations on their reactions with special reference to daily visiting. *Lancet*, *ii*, 1257–1262.

Joint Committee of the Royal College of Obstetricians and Gynaecologists and the Population Investigation Committee (1948) *Maternity in Great Britain*. London: Oxford University Press.

Knobloch, H., Rider, R., Harper, P., *et al* (1956) Neuropsychiatric sequelae of prematurity. *Journal of the American Medical Association*, **161**, 581–585.

Kuh, D. & Wadsworth, M E. J. (1989) Parental height, childhood environment and subsequent adult height in a national birth cohort. *International Journal of Epidemiology*, **18**, 663–668.

Langner, T. S. & Michael, S. (1963) *Life Stress and Mental Health*. Glencoe, Illinois: Free Press.

Lloyd, C. (1980) Life events and depressive disorder reviewed: I. Events as predisposing factors. *Archives of General Psychiatry*, **37**, 529–535.

MacMahon, B & Pugh, T. F. (1970) *Epidemiology: Principles and Methods*. Boston: Little, Brown.

Marris, P. (1958) *Widows and their Families*. London: Routledge & Kegan Paul.

Munro, A. (1966) Parental deprivation in depressive patients. *British Journal of Psychiatry*, **112**, 443–459

—— & Griffiths, A B (1969) Some psychiatric non-sequelae of childhood bereavement. *British Journal of Psychiatry*, **115**, 305–311.

Newton, G & Levine, S. (1968) *Early Experience and Behavior: The Psychobiology of Development*. Springfield, Illinois: Thomas.

Parker, G. (1979) Parental characteristics in relation to depressive disorders. *British Journal of Psychiatry*, **134**, 138–147.

—— (1983) Parental 'affectionless control' as an antecedent to adult depression. *Archives of General Psychiatry*, **40**, 956–960.

Parnas, J., Schulsinger, F., Teasdale, T. W., *et al* (1982) Perinatal complications and clinical outcome within the schizophrenia spectrum. *British Journal of Psychiatry*, **140**, 416–420.

Paykel, E. S. (1982) Life events and early environment. In *Handbook of Affective Disorders* (ed E. S. Paykel). Edinburgh: Churchill Livingstone.

Perris, C., Holmgren, S., Von Knorring, L., *et al* (1986) Parental loss by death in the early childhood of depressed patients and of their healthy siblings. *British Journal of Psychiatry*, **148**, 165–169.

Pidgeon, D. A. (1964) Tests used in the 1954 and 1957 surveys. In *The Home and the School*. London: MacGibbon and Kee.

—— (1968) Details of the fifteen year tests. In *All Our Future* (eds J. W. B. Douglas, J. M. Ross & H. R. Simpson). London· Peter Davies

Pless, I. B. (1984) Clinical assessment: physical and psychological functioning. In *Paediatric Clinics of North America*, **31**, 33–45.

Power, C., Fogelman, K. & Fox, A. J (1986) Health and social mobility during the early years of life. *Quarterly Journal of Social Affairs*, **2**, 397–413.

Prugh, D. G., Staub, E. M., Sands, H H., *et al* (1953) A study of the emotional reactions of children and families to hospitalization and illness. *American Journal of Orthopsychiatry*, **23**, 70–106.

Quinton D. & Rutter, M. (1976) Early hospital admissions and later disturbances of behavior: an attempted replication of Douglas' findings. *Developmental Medicine and Child Neurology*, **18**, 447–459.

Ragan, P. V. & McGlashan, T. H. (1986) Childhood parental death and adult psychopathology. *American Journal of Psychiatry*, **143**, 153–157.

Robins, L. N. & Ratcliff, K. S. (1980) The long-term outcome of truancy. In *Out of School* (eds L. Hersov & I. Berg). Chichester: John Wiley.

Rodgers, B (1978) Feeding in infancy and later ability and attainment: a longitudinal study. *Developmental Medicine and Child Neurology*, **20**, 421–426.

—— (1979) The prospects for ESN(M) school leavers *Special Education*, **6**, 8–9.

—— (1986) Change in the reading attainment of adults: a longitudinal study. *British Journal of Developmental Psychology*, **4**, 1–17.

—— (1990*a*) Behaviour and personality in childhood as predictors of adult psychiatric disorder. *Journal of Child Psychology and Psychiatry*, **31**, 393–414.

—— (1990*b*) Influences of early-life and recent factors on affective disorder in women: an exploration of vulnerability models. In *Straight and Devious Pathways from Childhood to Adulthood* (eds L. N. Robins & M. Rutter). New York: Cambridge University Press

—— & Mann, S. A. (1986) The reliability and validity of PSE assessments by lay interviewers: a national population survey *Psychological Medicine*, **16**, 689–700.

Roff, M. (1974) Childhood antecedents of adult neurosis, severe bad conduct and psychological health. In *Life History Research in Psychopathology*, vol 3 (eds D. F. Ricks, A. Thomas & M. Roff). Minneapolis: University of Minnesota Press.

Rogers, M. E., Lilienfeld, A. M. & Pasamanick, B (1955) Prenatal and paranatal factors in the development of childhood behavior disorders. *Acta Psychiatrica et Neurologica Scandinavica* (suppl. 102), 1–157.

Roy, A. (1983) Early parental death and adult depression *Psychological Medicine*, **13**, 861–865.

Rutter, M. (1981) *Maternal Deprivation Reassessed* (2nd edn) Harmondsworth: Penguin.

RUTTER, M. (1983) Statistical and personal interactions: facets and perspectives. In *Human Development: An Interactional Perspective* (eds D. Magnusson & V. L. Allen). New York: Academic Press.

—— (1984) Continuities and discontinuities in socioemotional development: empirical and conceptual perspectives. In *Continuities and Discontinuities in Development* (eds R. N. Emde & R. J Harman). New York· Plenum.

—— & GRAHAM, P (1970) Psychiatric aspects of intellectual and educational retardation. In *Education, Health and Behaviour* (eds M. Rutter, J. Tizard & K. Whitmore). London: Longman.

—— & QUINTON, D. (1984) Parental psychiatric disorder: effects on children. *Psychological Medicine*, **14**, 853–880.

SAUNDERS, W. A. & BARKER, M. G. (1972) Dyslexia as a cause of psychiatric disorder in adults. *British Medical Journal*, *iv*, 759–761.

SETHI, B. B. (1964) Relationship of separation to depression. *Archives of General Psychiatry*, **10**, 486–496.

SLATER, E. (1962) Birth order and maternal age of homosexuals. *Lancet*, *i*, 69–71.

STEIN, Z. & SUSSER, M. (1960) The families of dull children: a classification for predicting careers. *British Journal of Preventive and Social Medicine*, **14**, 83–88.

TENNANT, C., BEBBINGTON, P. & HURRY, J. (1980) Parental death in childhood and risk of adult depressive disorders: a review. *Psychological Medicine*, **10**, 289–299.

TIZARD, J. (1974) Longitudinal studies: problems and findings. In *Mental Deficiency: The Changing Outlook* (3rd edn) (eds A. M. Clarke & A. D. B. Clarke). London: Methuen.

TYRER, P. & TYRER, S. (1974) School refusal, truancy and adult neurotic illness. *Psychological Medicine*, **4**, 416–421.

WADSWORTH, M. E. J (1979) *Roots of Delinquency: Infancy, Adolescence and Crime*. Oxford: Martin Robertson.

—— (1986) Serious illness in childhood and its association with later-life achievement. In *Class and Health* (ed. R. G. Wilkinson). London: Tavistock.

—— (1987) Follow-up of the first national birth cohort: findings from the Medical Research Council National Survey of Health and Development. *Paediatric and Perinatal Epidemiology*, **1**, 95–117.

—— & FREEMAN, S. R. (1983) Generation differences in beliefs· a cohort study of stability and change in religious beliefs. *British Journal of Sociology*, **34**, 416–437.

—— & MACLEAN, M. (1986) Parents' divorce and children's life chances. *Children and Youth Services Review*, **8**, 145–159.

WARR, P. (1987) *Work, Unemployment and Mental Health* Oxford: Oxford University Press.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *Present State Examination*. London: Cambridge University Press.

——, NIXON, J. M , MANN, S. A., *et al* (1977) Reliability of the PSE (ninth edition) used in a population study. *Psychological Medicine*, **7**, 505–516.

——, MANN, S. A., LEFF, J. P., *et al* (1978) The concept of a 'case' in psychiatric population surveys. *Psychological Medicine*, **8**, 203–217.

—— & STURT, E. (1978) *The PSE-ID-CATEGO System: Supplementary Manual*. London: Institute of Psychiatry (mimeo)

Bryan Rodgers, PhD, *MRC National Survey of Health and Development, University College London and The Middlesex Hospital Medical School, Department of Community Medicine, 66–72 Gower Street, London WC1E 6EA*

British Journal of Psychiatry (1990), 157, 551–557

Hormonal Responses to Fenfluramine in Depressive Subtypes

PHILIP MITCHELL, GEORGE SMYTHE, GORDON PARKER, KAY WILHELM, IAN HICKIE, HENRY BRODATY and PHILIP BOYCE

In order to study putative differences in central neurotransmitter function in depressive subtypes, serum cortisol and prolactin responses to the putative serotonin agonist fenfluramine were examined in 30 subjects with major depression. Patients with endogenous depression (melancholia) as defined by each of ICD-9, DSM-III, RDC and Newcastle scale demonstrated a reduced prolactin response to 60 mg oral fenfluramine when compared with non-endogenous subjects. This was independent of either prolactin or cortisol baseline levels, and indicates that there are differences in brain neurotransmitter function in the endogenous and non-endogenous subtypes of depression. Basal prolactin levels were reduced in bipolar compared with unipolar subjects, and delusional compared with non-delusional patients, although there were no differences in the prolactin responses to fenfluramine between these subgroups. Basal cortisol levels and cortisol response to fenfluramine did not distinguish between any of the subtypes.

Since at least the 1920s (Gillespie, 1929) the subclassification of depression has been debated (Farmer & McGuffin, 1989). As noted recently, "an assumption of psychiatric research is that there is at least one form of depression that constitutes a disease with an underlying pathophysiologic dysfunction" (Hsiao *et al*, 1989), although it remains controversial as to whether the endogenous/melancholic 'disease' differs from residual disorders as a distinct type of depression or by severity.

To demonstrate the validity of subtypes, a number of approaches have been adopted: first, a search for necessary and sufficient clinical features (Foulds, 1973); second, multivariate statistical techniques such as factor analysis, discriminant analysis, cluster analysis and latent-class analysis; and third, biological markers such as the dexamethasone suppression test (Andreasen, 1982). Despite early enthusiastic claims (e.g. Carroll *et al*, 1981), there is no clinically or statistically derived system, or biological marker, accepted as yet as a 'gold standard'.

More recently, hormonal responses to pharmacological challenges have been used to examine central neurotransmitter function in depression indirectly and so indicate possible subtypes (Checkley, 1980). Differences between clinically diagnosed endogenous and non-endogenous depressive patients have been demonstrated with challenges such as the response of growth hormone (GH) to clonidine (Matussek *et al*, 1980; Checkley *et al*, 1984) and the cortisol response to methylamphetamine (Checkley, 1979), suggesting the possibility of differences in central noradrenergic activity.

Following findings such as reduced levels of the serotonin metabolite 5-HIAA in the cerebrospinal fluid (CSF) of depressives (Van Praag *et al*, 1970), hormonal responses to putative serotonergic challenges have been studied to examine the hypothesis of diminished serotonin function in depression. Abnormal responses to 5-hydroxytryptophan (5-HTP) (Meltzer *et al*, 1984), *l*-tryptophan (Heninger *et al*, 1984; Cowen & Charig, 1987) and *d,l*-fenfluramine (Siever *et al*, 1984; Weizman *et al*, 1988; Coccaro *et al*, 1989) have also been interpreted as indicating reduced serotonergic activity.

These challenges have additionally been assessed as possible discriminators of depressive subtypes. Cowen & Charig (1987) reported the prolactin response to *l*-tryptophan as reduced in melancholic subjects, after exclusion of those with a weight loss of greater than 10 lb. Lopez-Ibor *et al* (1988, 1989) reported a significant negative correlation between a measure of 'endogeneity' – the Newcastle scale score (Carney *et al*, 1965) – and increase in prolactin after fenfluramine administration, as well as demonstrating a difference in this response between subjects with DSM-III major depression with melancholia and those with dysthymic disorder (American Psychiatric Association, 1980). They did not, however, establish any difference between melancholic and non-melancholic patients with major depression.

There have been few comparisons of other clinical subtypes of depression. Meltzer *et al* (1984), using 5-HTP, found a reduced cortisol response in psychotic compared with non-psychotic subjects, while Maes *et al* (1987), using the same challenge, found an increased cortisol response in subjects with major compared with minor depression according to Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978). The only comparison of bipolar and unipolar depressed subjects found no difference in the cortisol response to 5-HTP (Meltzer *et al*, 1984).

Intercorrelations of the hormonal responses to these challenges with features of depression (such as suicidal ideation) postulated to be due to serotonergic dysfunction have also been examined. The majority of studies have found no relationship between such responses and severity of depression. They have, however, been demonstrated to correlate with both suicidal ideation (Meltzer *et al*, 1984; Cowen & Charig, 1987) and weight loss (Cowen & Charig, 1987). Coccaro *et al* (1989) reported a reduced prolactin response to fenfluramine in depressed and personality-disordered patients who had attempted suicide compared with those who had not, irrespective of diagnosis.

We have focused on hormonal responses to fenfluramine as an index of central neurotransmitter function. Because of concerns about reliability of these responses (Van Praag *et al*, 1987), we carried out a single-blind, placebo-controlled study in 14 normal subjects which demonstrated a significant release of prolactin and cortisol ($P<0.01$), but not of growth hormone, in response to fenfluramine; we then investigated 27 depressed and 14 control subjects, establishing elevated baseline levels of cortisol and reduced baseline levels of prolactin in the depressed group (Mitchell & Smythe, 1990). Although the prolactin response in endogenous/melancholic subjects was reduced compared with controls, this was partially dependent upon abnormal baseline levels. There was no abnormality of the cortisol response to fenfluramine.

In this study, of an expanded sample of 30 depressed patients, we examined prolactin and cortisol responses to fenfluramine in clinically diagnosed endogenous/non-endogenous, unipolar/bipolar and delusional/non-delusional depressives to investigate central neurotransmitter differences between variously defined depressive subtypes. We also examined for associations between these hormonal responses and specific features of depression, such as suicidal ideation and weight disturbance.

Method

The 30 depressed subjects all met DSM-III criteria for major depression, derived from a semistructured interview (Brodaty *et al*, 1987). Because of recent evidence indicating that the number of patients allocated to depressive subtypes varies significantly depending on the classification system (Davidson *et al*, 1984), subjects were classified according to each of the following diagnostic systems: DSM-III, RDC, ICD-9 (World Health Organization, 1978) and the Newcastle scale. For the RDC system, subjects were divided into endogenous ('definite' endogenous) and non-endogenous ('probable' endogenous plus non-endogenous) subtypes to examine a more clearly identified endogenous group. Likewise for ICD-9, subjects were allocated to endogenous (296.1, 296.3) and non-endogenous (300.4) subgroups. With the Newcastle scale a score of six or more was required for the diagnosis of endogenous depression. Subjects with significant medical disorders or regular alcohol intake exceeding 30 g daily were excluded. One provisional subject with a baseline prolactin level of 2963 mIU/l was excluded from the analyses, reducing the original sample of 31 subjects. This subject later admitted to having miscarried several days before the test. All subjects gave written informed consent to the procedure. A relatively small number of subjects were defined as endogenous depressives by the Newcastle scale, but this reflects the tendency of our research group to rate this scale conservatively.

Subjects were required to have been free of antidepressant or other psychotropic medication for at least two weeks, although benzodiazepine hypnotics were allowed. Three subjects had been tested with intravenous clonidine 2–19 days before administration of fenfluramine.

Severity of depression was assessed by both the observer-rated 21-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) and the Zung self-rating scale (Zung, 1965). Weight loss during the current depressed episode was assessed using an item from the semistructured interview (1 = no weight loss, 2 = <3 kg; 3 = 3–5 kg; 4 = >5 kg). Suicidal ideation was measured using items from the Hamilton and Zung scales, and the semistructured interview.

Subjects fasted from midnight and had a catheter inserted into an antecubital vein between 8 a.m. and 8:30 a.m., after which they rested for one hour. Three baseline blood samples were taken at quarter-hourly intervals, then 60 mg *d,l*-fenfluramine was administered orally. Blood samples were then drawn hourly for five hours while the subject remained supine. A light meal was provided four hours after fenfluramine administration. Blood samples were centrifuged at 4 °C for 10 minutes at 1000g within one hour of collection and the serum stored at −20 °C.

Serum prolactin was measured using a double antibody radioimmunoassay, with an intra-assay coefficient of variation (c.v.) of 5% and inter-assay c.v. of 6.5% at 658 mIU/l. Serum cortisol was measured by radioimmunoassay of the total cortisol with an intra-assay c.v. of 6.8% and an interassay c.v. of 7.9% at 375 nmol/l.

Baseline concentrations of prolactin and cortisol were taken as the average of the three pre-fenfluramine levels. Hormonal responses were analysed as the absolute increase to peak above baseline (Δ) and the area under the response curve (AUC). The frequency distributions of the examined variables were assessed by means of the Shapiro & Wilk test for normality of data (Madansky, 1988). For normally distributed data (baseline cortisol levels only), Student's *t*-test was used to compare group means. For non-normally distributed data, variables were $\log_{10}$-transformed before use of *t*-tests. Analysis of covariance (ANCOVA) was used to determine the effect of covariates. Spearman's rank-order correlations were used to assess relationships between variables.

Results

Baseline cortisol levels (Fig. 1) did not differ significantly between subgroups using any of the diagnostic systems (Table I). For cortisol, Δ and the AUC correlated significantly ($r=0.87$, $P<0.001$), but there were no significant differences between groups on either (Table II).

Baseline prolactin levels (Fig. 2) were either reduced significantly or showed a trend towards reduction using ICD-9 ($t=-2.00$, $P=0.06$), DSM-III ($t=-1.95$, $P=0.06$) and RDC ($t=-2.43$, $P<0.05$) definitions of endogenous as against non-endogenous subjects (Table I). After covarying for age, however, there were no significant differences between these groups. For prolactin, Δ and AUC correlated significantly ($r=0.93$, $P<0.001$). The Δ values were reduced in the endogenous compared with non-endogenous depressed subjects as defined by each of the four classificatory systems (Table II). The AUC was correspondingly significantly reduced in ICD-9 and RDC endogenous subjects.

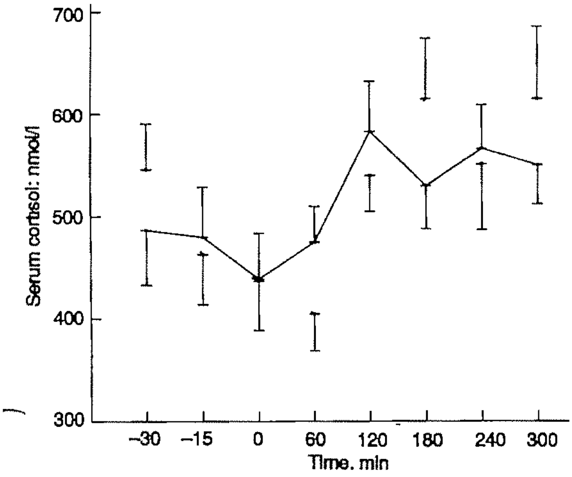


FIG. 1 Cortisol response to fenfluramine (±s.e.m.) in ICD-9 endogenous (———) and non-endogenous (·· ···· ····) subgroups

ANCOVA was then undertaken with a series of covariates to identify other factors which may have accounted for the observed differences in prolactin Δ values between groups. They remained after covarying for sex ($P<0.05$), and as indicated in Table III, significant differences or strong trends towards significance also remained across all diagnostic systems after covarying for age, severity (as measured by Zung or Hamilton scores), weight loss, baseline prolactin, and baseline cortisol levels.

The most significant difference between subgroups was in the ICD-9-defined subjects, in which reduction of prolactin Δ values in endogenous compared with non-endogenous subjects remained significant after covarying for each of the above variables individually. The difference remained significant ($F=4.56$, d.f. $=1,21$, $P<0.05$) even when covarying for all the following variables in combination: age, sex, Zung score, weight loss, prolactin and cortisol baseline values. A strong trend towards significance ($F=3.65$, d.f. $=1,22$, $P=0.07$) also persisted when the Hamilton, rather than Zung, severity score was used in ANCOVA with the other covariates.

The prolactin AUC was also still reduced significantly in ICD-9 endogenous subjects after covarying for each of the above covariates individually ($P<0.05$). There were, however, no significant differences in the prolactin AUC between endogenous and non-endogenous subjects defined by the other classification systems after covarying for these variables.

There were only four bipolar and five delusionally depressed subjects in the sample of 30. There were, however, significantly lower baseline prolactin levels in the bipolar compared with unipolar subjects ($t=3.01$, $P<0.01$), even after covarying for age ($F=10.03$, d.f. $=2,27$, $P<0.01$). Similarly, baseline prolactin was reduced in delusional compared with non-delusional subjects ($t=2.29$, $P<0.05$), with a strong trend towards significance after covarying for age ($F=3.50$, d.f. $=2,27$, $P=0.07$). There were no differences between these groups in the prolactin responses to fenfluramine, nor in the cortisol baseline levels or responses.

Prolactin and cortisol responses did not correlate significantly with severity of depression as rated by either total Hamilton or Zung scores. For example, there were no significant intercorrelations between prolactin Δ values and the Hamilton ($r=-0.12$) or Zung ($r=-0.06$) scores. The only significant association between hormone responses and measurements of suicidal ideation was a weak positive

TABLE I

Raw mean (s.e.m.) cortisol and prolactin baseline levels in endogenous and non-endogenous depressives

	ICD-9	*DSM-III*	*RDC*	*Newcastle scale*
Cortisol: nmol/l				
endogenous	469 (48)	486 (42)	480 (38)	516 (77)
non-endogenous	482 (42)	463 (49)	468 (57)	463 (35)
Prolactin: mIU/l				
endogenous	133 (18)*	135 (17)*	133 (16)**	143 (31)
non-endogenous	180 (20)	182 (21)	192 (23)	161 (16)

*$P<0.10$, **$P<0.05$, t-tests after $\log_{10}$-transformation.

TABLE II

Raw mean (s.e.m.) cortisol and prolactin responses to fenfluramine in endogenous and non-endogenous depressives

	No. of subjects	*Age: years*	*Cortisol: nmol/l* Δ	*Cortisol: nmol/l* *Area under curve*	*Prolactin: mIU/l* Δ	*Prolactin: mIU/l* *Area under curve*
ICD-9						
endogenous	15	51.8 (3.9)*	197 (27)	181 (66)	230 (33)**	299 (54)*
non-endogenous	15	37.7 (3.7)	220 (46)	166 (75)	455 (79)	506 (87)
DSM-III						
melancholic	16	50.3 (3.9)*	203 (27)	171 (62)	248 (31)*	317 (49)
non-melancholic	14	38.4 (4.0)	215 (49)	177 (81)	451 (87)	501 (97)
RDC						
endogenous	18	50.8 (4.0)**	203 (24)	187 (55)	250 (29)*	331 (45)*
non-endogenous	12	35.6 (2.8)	216 (57)	153 (94)	482 (98)	501 (112)
Newcastle scale						
endogenous	7	57.4 (6.4)*	136 (30)	13 (82)[1]	195 (31)*	249 (42)
non-endogenous	23	40.9 (2.9)	230 (32)	222 (56)	387 (57)	240 (66)

*$P<0.05$, **$P<0.01$, t-tests after $\log_{10}$-transformation.

[1] Range −313 to 196.

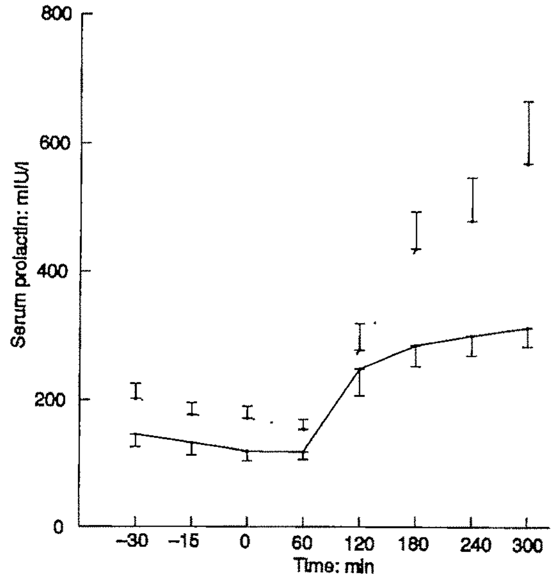


FIG.2 Prolactin response to fenfluramine (±s.e.m.) in ICD-9 endogenous (————) and non-endogenous (············) subgroups.

correlation between Δ for prolactin and score on the Hamilton suicide item ($r=0.41$, $P<0.05$).

In light of the findings of Cowen & Charig (1987), we compared those with more than 5 kg weight loss against those with less than 5 kg weight loss during the episode of depression. Baseline cortisol levels were significantly elevated in those who lost more weight (643 nmol/l v. 433 nmol/l; $t=3.01$, $P<0.01$). Although the cortisol AUC was also lower in this group ($t=-2.07$, $P<0.05$), ANCOVA demonstrated that there was an important effect of baseline cortisol levels ($F=0.49$, d.f. = 1,27, $P=0.49$). While baseline prolactin levels were not different, there was a trend ($t=-1.77$, $P=0.09$) towards a reduced Δ in those who lost more weight that was independent of baseline cortisol ($F=4.05$, d.f. = 1,27, $P=0.05$).

We also compared those not taking benzodiazepine with those who were, as Nutt & Cowen (1987) have demonstrated that acute but not chronic benzodiazepine use reduces hormonal responses to *l*-tryptophan. We found no significant differences (or even trends) in baseline levels or responses of either hormone.

Discussion

This is the first definitive demonstration of a reduced prolactin response to fenfluramine in endogenous compared with non-endogenous subjects with major depression. Although Lopez-Ibor *et al* (1988, 1989) reported suggestive evidence, they only found a significant difference between DSM-III major depression with melancholia and dysthymic disorder. Our demonstration of a reduced response in endogenous subjects is consistent with the finding of Shapira *et al* (1989) of an increase in the fenfluramine-induced prolactin levels in 10 RDC-defined endogenous subjects with major depression after three weeks of treatment with imipramine. Our finding is also consistent with the report of Cowen & Charig (1987), who demonstrated a diminished prolactin response to *l*-tryptophan in their melancholic subjects.

The validity of our finding was supported by the demonstration that there was a significant reduction of prolactin Δ values in the endogenous compared with non-endogenous subjects defined by each of ICD-9, DSM-III, RDC and Newcastle scale. The prolactin AUC was also significantly lower in ICD-9 and RDC-defined endogenous subjects.

TABLE III
Effect of covariates on comparisons of prolactin Δ values between endogenous and non-endogenous subgroups

		ANCOVA with single covariate					
	t-test	*Age*	*Zung score*	*Hamilton score*	*Cortisol baseline*	*Prolactin baseline*	*Weight loss*
ICD-9	$t=-3.09$	$F=7.33$	$F=9.20$	$F=7.53$	$F=9.22$	$F=6.88$	$F=6.56$
	$P<0.01$	$P=0.01$	$P<0.01$	$P=0.01$	$P<0.01$	$P=0.01$	$P<0.05$
DSM-III	$t=-2.24$	$F=3.50$	$F=4.65$	$F=3.32$	$F=4.86$	$F=3.21$	$F=3.19$
	$P<0.05$	$P=0.07$	$P<0.05$	$P=0.08$	$P<0.05$	$P=0.09$	$P=0.09$
RDC	$t=-2.45$	$F=4.15$	$F=5.60$	$F=4.29$	$F=5.79$	$F=3.62$	$F=3.25$
	$P<0.05$	$P=0.05$	$P<0.05$	$P<0.05$	$P<0.05$	$P=0.07$	$P=0.08$
Newcastle scale	$t=-2.19$	$F=3.17$	$F=4.78$	$F=3.16$	$F=4.71$	$F=4.31$	$F=3.04$
	$P<0.05$	$P=0.09$	$P<0.05$	$P=0.09$	$P<0.05$	$P<0.05$	$P=0.09$

A particular strength of our study was the testing for relevant covariates that may have accounted for intergroup differences. This has not been undertaken in many of the previous studies examining for biological distinctions between depressive subtypes. Using all four classificatory systems, the reduction in prolactin Δ values in endogenous compared with non-endogenous subjects continued to be either significant, or demonstrated a strong trend towards significance, after covarying individually for age, sex or depression severity (as measured by Hamilton or Zung scores). These factors are often significant covariates in determining hormonal responsiveness (Checkley, 1980). Unlike Cowen & Charig (1987), we did not find an effect of weight loss. However, this is in keeping with a recent study from the same group (Anderson *et al*, 1989) which demonstrated no effect of moderate weight loss in normal subjects on either sleep-related prolactin secretion or prolactin responses to metoclopramide or thyrotrophin-releasing hormone.

Although the reduced prolactin response in endogenous depressed compared with control subjects is partially dependent on baseline prolactin and cortisol levels (Mitchell & Smythe, 1990), we have found in the present study that the difference between endogenous and non-endogenous subjects is independent of these covariates.

Using the traditional ICD-9 system, the difference between subgroups was significant even after covarying for each of the covariates in combination, except with the Hamilton Scale, for which there was still a strong trend towards significance. The latter is, however, an index of both severity and typology (Joyce & Paykel, 1989) as it includes a number of items loading for endogenous depression, such as guilt and paranoid ideation. This confounding of severity and typology is a critical issue when examining for potential differences between endogenous and non-endogenous subgroups.

The finding that the differences in prolactin response were more significant using the ICD-9 classification system is of particular interest. It is consistent with the view put forward by Carroll (1989) that, although the ICD-9 system is not operationalised and therefore perhaps less reliable, it may represent a more valid diagnostic system than others such as DSM-III and RDC as it "follows the historical, European, Kraepelinian concepts of manic depressive illness and melancholia". The diagnostic guidelines for the original studies of the dexamethasone suppression test comparing endogenous and non-endogenous subjects were based upon the ICD-8 and ICD-9 glossaries (Carroll *et al*, 1980).

Although we studied several measurements of suicidal ideation, we only found an incongruous positive association between suicidal intent and prolactin Δ values. With regard to the other subclassifications of depression, we demonstrated a reduction in basal prolactin levels in bipolar compared with unipolar, and delusional compared with non-delusional depressed subjects. Although there were only four bipolar patients in the sample of 30, the finding is of interest as it is consistent with the reports of both Mendlewicz *et al* (1980) and Joyce *et al* (1988). Baumgartner *et al* (1988) found no difference between their unipolar and bipolar subjects. To our knowledge this is the first demonstration of reduced prolactin levels in delusional compared with non-delusional depressed subjects. Lykouras *et al* (1988) found no difference in prolactin levels between these subgroups.

It should be borne in mind that although fenfluramine is usually considered to act on the serotonergic system, there is no conclusive evidence that the effects of this drug are mediated via a highly selective action on one specific neurotransmitter (Van Praag *et al*, 1987). While there is some evidence to suggest that serotonin mediates the prolactin response to fenfluramine (Willoughby *et al*, 1982; Quattrone *et al*, 1983; Van de Kar *et al*, 1985) and perhaps pituitary ACTH release (Barbieri *et al*, 1984; Lewis & Sherman 1984; Van de Kar *et al*, 1985), recent animal studies, however, have indicated that the effects of fenfluramine on pituitary ACTH and prolactin release may be mediated by central catecholaminergic rather than serotonergic pathways (Smythe *et al*, 1982; Storlien & Smythe, 1990). These findings are supported by other data showing that, in man also, the release of pituitary ACTH by fenfluramine is mediated by central alpha-2 adrenoceptors (Jackson *et al*, 1988). The significance of the findings of this present study with respect to central neurotransmitter activity in depressive subtypes must therefore remain uncertain until the actual interaction of fenfluramine with the different neurotransmitters is clearly ascertained.

In summary, the findings of our study indicate that the prolactin response to fenfluramine differs between endogenous and non-endogenous subjects, pointing to differences in brain neurotransmitter function in depressives being revealed by this challenge, and supporting the clinical distinction between different subtypes of depressive disorders.

Acknowledgements

This research was supported by a grant from the Australian National Health & Medical Research Council. We thank Mr Dusan Hadzi-Pavlovic for statistical advice, Ms Christine Burke and Ms Anna Zournazi for their technical assistance, and Mrs Zora Vuckovic for preparation of the manuscript.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Anderson, I. M., Crook, W. S., Garside, S. E., *et al* (1989) Effect of moderate weight loss on prolactin secretion in normal female volunteers. *Psychiatry Research*, **29**, 161–167.

Andreasen, N. C. (1982) Concepts, diagnosis and classification. In *Handbook of Affective Disorders* (ed. E. S. Paykel), pp. 24–44. Edinburgh: Churchill Livingstone.

Barbieri, C., Bigatti, G., Rauhe, W. G., *et al* (1984) Serotonergic regulation of cortisol secretion in dogs. *Endocrinology*, **115**, 748–751.

Baumgartner, A., Graef, K-J. & Kuerten, I. (1988) Prolactin in patients with major depressive disorders and in healthy subjects: 1. Cross-sectional study of basal and post-TRH and post-dexamethasone prolactin levels. *Biological Psychiatry*, **24**, 249–267.

Brodaty, H., Boyce, P., Wilhelm, K., *et al* (1987) The establishment of a mood disorders unit. *Australian and New Zealand Journal of Psychiatry*, **21**, 375–382.

Carney, M. W. P., Roth, M. & Garside, R. F. (1965) The diagnosis of depressive symptoms and the prediction of ECT response. *British Journal of Psychiatry*, **111**, 639–645.

Carroll, B. J. (1989) Diagnostic validity of laboratory studies: rules of the game. In *The Validity of Psychiatric Diagnosis* (eds L. Robins & J. E. Barrett). New York: Raven Press.

——, Feinberg, M., Greden, J. F., *et al* (1980) Diagnosis of endogenous depression: comparison of clinical research and neuroendocrine criteria. *Journal of Affective Disorders*, **2**, 177–194.

——, ——, ——, *et al* (1981) A specific laboratory test for the diagnosis of melancholia. Standardization, validation and clinical utility. *Archives of General Psychiatry*, **38**, 15–22.

Checkley, S. A. (1979) Corticosteroid and growth hormone responses to methylamphetamine in depressive illness. *Psychological Medicine*, **9**, 107–115.

—— (1980) Neuroendocrine tests for monoamine function in man: a review of basic theory and its application to the study of depressive illness. *Psychological Medicine*, **10**, 35–53.

——, Glass, I. B., Thompson, C., *et al* (1984) The GH response to clonidine in endogenous as compared with reactive depression. *Psychological Medicine*, **14**, 773–777.

Coccaro, E. F., Siever, L. J., Klar, H. M., *et al* (1989) Serotonergic studies in patients with affective and personality disorders: correlations with suicidal and impulsive aggressive behaviour. *Archives of General Psychiatry*, **46**, 587–599.

Cowen, P. J. & Charig, E. M. (1987) Neuroendocrine responses to intravenous tryptophan in major depression. *Archives of General Psychiatry*, **44**, 958–966.

Davidson, J., Turnbull, C., Strickland, R., *et al* (1984) Comparative diagnostic criteria for melancholia and endogenous depression. *Archives of General Psychiatry*, **41**, 506–511.

Dolan, R. H. & Calloway, S. P. (1986) The human growth hormone response to clonidine: relationship to clinical and neuroendocrine profile in depression. *American Journal of Psychiatry*, **143**, 772–774.

Farmer, A. & McGuffin, P. (1989) The classification of the depressions: contemporary confusion revisited. *British Journal of Psychiatry*, **155**, 437–443.

Foulds, G. A. (1973) The relationship between the depressive illnesses. *British Journal of Psychiatry*, **123**, 531–533.

Gillespie, R. D. (1929) The clinical differentiation of types of depression. *Guy's Hospital Reports*, **79**, 306–344.

Hamilton, M. (1967) Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, **6**, 278–296.

Heninger, G. R., Charney, D. S. & Sternberg, D. E. (1984) Serotonergic function in depression: prolactin response to intravenous tryptophan in depressed patients and healthy subjects. *Archives of General Psychiatry*, **41**, 398–402.

Horton, R. W., Katona, C. L. E., Theodorou, A. E., *et al* (1986) Platelet radioligand binding and neuroendocrine challenge tests in depression. In *Antidepressants and Receptor Function*, Ciba Foundation Symposium, No. 123, pp. 84–105. Chichester: Wiley.

Hsiao, J. K., Bartko, J. J. & Potter, W. Z. (1989) Diagnosing diagnoses: receiver operating characteristic methods and psychiatry. *Archives of General Psychiatry*, **46**, 664–667.

Jackson, R. V., Grice, J. E. & Jackson, A. J. (1988) Inhibition of serotonin-induced ACTH release in man by clonidine. *Clinical and Experimental Pharmacology and Physiology*, **15**, 293–298.

Joyce, P. R., Sellman, J. D., Donald, R. A., *et al* (1988) The unipolar-bipolar depressive dichotomy and the relationship between afternoon prolactin and cortisol levels. *Journal of Affective Disorders*, **14**, 189–193.

—— & Paykel, E. S. (1989) Predictors of drug response in depression. *Archives of General Psychiatry*, **46**, 89–99.

Lewis, D. A. & Sherman, B. M. (1984) Serotonergic stimulation of adrenocorticotropin secretion in man. *Journal of Clinical Endocrinology and Metabolism*, **58**, 458–462.

Lopez-Ibor, J. J., Saiz-Ruiz, J. & Iglesias, L. M. (1988) The fenfluramine challenge test in the affective spectrum: a possible marker of endogeneity and severity. *Pharmacopsychiatry*, **21**, 9–14.

——, —— & —— (1989) Neuroendocrine challenges in the diagnosis of depressive disorders. *British Journal of Psychiatry*, **154** (suppl. 4), 73–76.

Lykouras, E., Markianos, M., Malliaras, D., *et al* (1988) Neurochemical variables in delusional depression. *American Journal of Psychiatry*, **145**, 214–217.

Madansky, A. (1988) *Prescriptions for Working Statisticians*. New York: Springer-Verlag.

Maes, M., De Ruyter, M., Claes, R., *et al* (1987) The cortisol responses to 5-hydroxytryptophan, orally, in depressive inpatients. *Journal of Affective Disorders*, 13, 23–30.

Matussek, N., Ackenheil, M., Hippius, H., *et al* (1980) Effect of clonidine on growth hormone release in psychiatric patients and controls. *Psychiatry Research*, 2, 25–36.

Meltzer, H. Y., Umberkoman-Wiita, B., Robertson, A., *et al* (1984) Effect of 5-hydroxytryptophan on serum cortisol levels in major affective disorders: 1. Enhanced response in depression and mania. *Archives of General Psychiatry*, 41, 366–374

Mendlewicz, J., Van Cauter, E., Linkowski, *et al* (1980) The 24-hour profile of prolactin in depression. *Life Sciences*, 27, 2015–2024.

Mitchell, P. & Smythe, G. (1990) Hormonal responses to fenfluramine in depressed and control subjects *Journal of Affective Disorders*, 19, 43–56.

Nutt, D. J. & Cowen, P. J. (1987) Diazepam alters brain 5-HT function in man: implications for the acute and chronic effects of benzodiazepines. *Psychological Medicine*, 17, 601–607.

Quattrone, A., Tedeschi, G., Aguglia, U., *et al* (1983) Prolactin secretion in man: a useful tool to evaluate the activity of drugs on central 5-hydroxytryptaminergic neurones. Studies with fenfluramine. *British Journal of Clinical Pharmacology*, 16, 471–475.

Roy, A., Pickar, D., Linnoila, M., *et al* (1985) Plasma norepinephrine level in affective disorders. *Archives of General Psychiatry*, 42, 1181–1185.

——, ——, Douillet, P., *et al* (1986) Urinary monoamines and monoamine metabolites in subtypes of unipolar depressive disorder and normal controls. *Psychological Medicine*, 16, 541–546.

Shapira, B., Reiss, A , Kaiser, N., *et al* (1989) Effect of imipramine treatment on the prolactin response to fenfluramine and placebo challenge in depressed patients. *Journal of Affective Disorders*, 16, 1–4.

Siever, L. J., Murphy, D. L., Slater, S., *et al* (1984) Plasma prolactin changes following fenfluramine in depressed patients compared to controls: an evaluation of central serotonergic responsivity in depression. *Life Sciences*, 34, 1029–1039.

Smythe, G. A , Bradshaw, J. E , Cai, W. Y., *et al* (1982) Hypothalamic serotonergic stimulation of thyrotropin secretion and related brain-hormone and drug interactions in the rat. *Endocrinology*, 111, 1181–1191

Spitzer, R L., Endicott, J. & Robins, E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, 35, 773–782

Storlien, L H. & Smythe, G. A. (1990) *D*-fenfluramine effects on hypothalamic monoamine activities and their hormonal correlates. *Life Sciences* (in press).

Van de Kar, L D , Urban, J. H , Richardson, K. D., *et al* (1985) Pharmacological studies on the serotonergic and non-serotonin-mediated stimulation of prolactin and corticosterone secretion by fenfluramine. *Neuroendocrinology*, 41, 283–288.

Van Praag, H. M., Korf, J. & Puite, J. (1970) 5-hydroxyindoleacetic acid levels in the cerebrospinal fluid of depressive patients treated with probenecid. *Nature*, 225, 1259–1260

——, Lemus, C & Kahn, R. (1987) Hormonal probes of central serotonergic activity· do they really exist? *Biological Psychiatry*, 22, 86–98.

Weizman, A., Mark, M , Gil-Ad, I , *et al* (1988) Plasma cortisol, prolactin, growth hormone and immunoreactive *B*-endorphin response to fenfluramine challenge in depressed patients. *Clinical Neuropharmacology*, 11, 250–256.

Willoughby, J., Menadue, M. & Jervois, P (1982) Function of serotonin in physiological secretion of growth hormone and prolactin and p-clorophenylalanine. *Brain Research*, 249, 291–299.

World Health Organization (1978) *Mental Disorders. Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Disease* (ICD-9) Geneva: WHO.

Zung, W. W. K. (1965) A self-rated depression scale. *Archives of General Psychiatry*, 12, 63–70.

*Philip Mitchell, MB, BS, MRCPsych, FRANZCP, *Senior Lecturer, School of Psychiatry, University of New South Wales, Sydney*; George Smythe, ASTC, BSc, PhD, *Head, C. E. Heath Neuroscience Laboratory, Garvan Institute of Medical Research, Sydney, and Senior Lecturer (Conjoint), University of New South Wales*; Gordon Parker, MD, PhD, FRANZCP, *Professor, School of Psychiatry, University of New South Wales*; Kay Wilhelm, MB, BS, FRANZCP, *Senior Staff Specialist, Division of Psychiatry, Prince Henry Hospital, and Lecturer (Conjoint), University of New South Wales*; Ian Hickie, MB, BS, FRANZCP, *Staff Specialist, Division of Psychiatry, Prince Henry Hospital, and Lecturer (Conjoint), University of New South Wales*; Henry Brodaty, MD, FRACP, FRANZCP, *Senior Staff Specialist, Division of Psychiatry, Prince Henry Hospital, and Senior Lecturer (Conjoint), School of Psychiatry, University of New South Wales*; Philip Boyce, MB, BS, FRANZCP, *Senior Lecturer, School of Psychiatry, University of New South Wales*

*Correspondence: *Mood Disorders Unit, Prince Henry Hospital, Little Bay, NSW, 2036 Australia*

British Journal of Psychiatry (1990), **157**, 558–561

Syndromes of Chronic Schizophrenia

PETER F. LIDDLE and THOMAS R. E. BARNES

Confirmation is reported of an earlier finding that the symptoms of patients with chronic schizophrenia segregate into three syndromes: psychomotor poverty (poverty of speech, flatness of affect, decreased spontaneous movement); disorganisation (disorders of the form of thought, inappropriate affect); and reality distortion (delusions and hallucinations).

Although schizophrenia is clinically heterogeneous, attempts to identify distinct types of schizophrenia have been only partially successful. For example, the classic subdivision into paranoid, hebephrenic, catatonic and simple schizophrenia (Bleuler, 1911) proves to be unsatisfactory because many patients exhibit characteristics of more than one of these subtypes. Similar problems have beset subsequent attempts. This difficulty suggests that the heterogeneity of schizophrenia is better described in terms of syndromes which can coexist in a patient.

It is possible that there is a single abnormality of brain function that is central to schizophrenia, and that this central abnormality can be associated with impaired function of several different regions or neuronal systems within the brain. Constitutional and environmental factors might determine the relative involvement of these different regions or systems. If this were the case, one would predict that the symptoms of schizophrenia would segregate into syndromes, such that the correlations between symptoms within each syndrome would be high, while the strength of correlations between symptoms from different syndromes would be expected to depend on the sample of patients studied.

The division of schizophrenic symptoms into positive and negative syndromes is an example of an attempt to describe the heterogeneity of schizophrenia in terms of distinguishable syndromes. For example, Crow (1980) proposed that type 1 schizophrenia, characterised by positive symptoms, and type 2 schizophrenia, characterised by negative symptoms, reflect independent dimensions of psychopathology, which are not mutually exclusive.

The positive–negative symptom dichotomy overlaps with the distinction between acute and chronic schizophrenia. Negative symptoms tend to be chronic, while positive symptoms are characteristic of acute schizophrenic episodes (Crow, 1980). This overlap between the positive–negative dichotomy and the distinction between acute and chronic schizophrenia presents difficulties for attempts to delineate the pathological processes associated with particular groups of symptoms, since differences between acute and chronic pathological processes might be confused with differences associated directly with symptom type.

In an attempt to circumvent this difficulty, Liddle (1987*a*,*b*) examined the segregation of schizophrenic symptoms, and the relationship between symptoms and neuropsychological performance, in a group of chronic schizophrenic patients who were relatively homogeneous with regard to chronicity. The symptoms in this group of chronic patients segregated into three syndromes, each associated with a particular pattern of performance on neuropsychological tests

TABLE I

*Syndromes of chronic schizophrenic symptoms previously identified by Liddle (1987*a,b*)*

Symptoms in each syndrome	*Impaired neuropsychological function in each syndrome*
Psychomotor poverty syndrome	
Poverty of speech	Conceptual reasoning
Decreased spontaneous movement	Long-term memory Object naming
Unchanging facial expression	
Paucity of expressive gesture	
Affective non-response	
Lack of vocal inflection	
Disorganisation syndrome	
Inappropriate affect	Concentration
Poverty of content of speech	New learning
Tangentiality	
Derailment	
Pressure of speech	
Distractibility	
Reality distortion syndrome	
Voices speak to the patient	Figure-ground perception
Delusions of persecution	
Delusions of reference	

(Table I). In this paper we report a replication of Liddle's study of the segregation of chronic schizophrenic symptoms in a different sample.

Method

The sample comprised all 57 patients satisfying DSM–III criteria for schizophrenia (American Psychiatric Association, 1980) resident in three wards for long-term patients at Horton Hospital, Epsom. There were 33 men and 24 women, aged 34–80 years (mean 58.6), with a mean duration of illness of 30.5 years (range 8–49). Fifty-four patients were receiving neuroleptic medication (mean daily dose in chlorpromazine equivalents 935 mg, range 72–4345). Thirty-nine patients were receiving anticholinergic medication.

Symptoms were assessed using the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982) and the Manchester scale (Krawiecka *et al*, 1977), by one of the investigators (TREB), who was blind to the details of the segregation of symptoms into three syndromes. In the previous study (Liddle, 1987*a*) symptoms were assessed according to Andreasen's Comprehensive Assessment of Symptoms and History, which includes SANS and also a comprehensive assessment of delusions, hallucinations and disorders of the form of thought. In this study the Manchester scale was used for the rating of delusions, hallucinations and incoherence of speech, because it is suitable for use with severely handicapped patients who cannot tolerate a long interview. Parkinsonism was assessed independently (by PFL) using Mindham's (1976) modified version of the Simpson and Angus scale. Demographic information and details of each patient's illness and treatment were collected from the case notes.

Items from SANS were selected for analysis according to the procedure used by Liddle (1987*a*). Thus items from the affective flattening and alogia subscales, rated 2 or more in at least 10% of the patients, were selected. In order to avoid making *a priori* assumptions about the relationships between items, the subscale global scores and the ratings of overall subjective experience of each of the subgroups were not included. The set of items selected from SANS, together with the Manchester scale scores, were subjected to factor analysis.

Initial factors were extracted by the method of principal factors, oblique rotation was performed, and factor scores were calculated by the regression method, using SPSS. Oblique rotation was performed because there is no *a priori* reason why factors representing different schizophrenic syndromes should be orthogonal. Pearson correlations between factor scores and age of onset of illness, length of illness, dose of antipsychotic medication, use of anticholinergic medications and severity of Parkinsonism were calculated, and statistical significance was calculated using two-tailed tests.

Results

Factor analysis yielded three factors which together accounted for 64.2% of the variance. The factor loadings and variance accounted for by each factor are shown in Table II. Despite allowing an oblique rotation, the factors

TABLE II
Factor loadings obtained by factor analysis of symptoms

Symptom	*Factor 1*	*Factor 2*	*Factor 3*
Psychomotor poverty syndrome (high loading on factor 1)			
Unchanging facial expression	0.92	−0.01	0.05
Affective non-response	0.89	0.03	0.16
Poverty of expressive gesture	0.87	−0.16	−0.00
Decreased spontaneous movements	0.86	−0.03	0.10
Lack of vocal inflections	0.86	0.03	0.07
Poverty of speech	0.71	−0.05	−0.38
Increased speech latency	0.62	0.23	−0.27
Poor eye contact	0.54	0.12	−0.37
Disorganisation syndrome (high loading on factor 2)			
Poverty of content of speech	0.04	0.69	0.19
Incoherence of speech	0.05	0.69	0.46
Inappropriate affect	−0.04	0.56	−0.18
Reality distortion syndrome (high loading on factor 3)			
Hallucinations	0.05	−0.04	0.68
Delusions	−0.13	0.22	0 67
% of total variance accounted for by each factor	41.0	12.1	16.9

are nearly orthogonal. There is a weak negative correlation ($r=0.25$) between factors 1 and 2, while the correlations between other pairs of factors are near zero. It should be noted that as a result of the departure from strict orthogonality, the total variance accounted for by the three factors is a little less than the sum of the percentages of variance accounted for by each factor separately.

Each symptom has a high loading on only one of the three factors. Thus the symptoms segregate into three syndromes, and the composition of each syndrome is virtually identical with the corresponding syndrome identified by Liddle (1987*a*), allowing for the lack of differentiation in the Manchester scale between different disorders of the form of thought, and different types of delusions and hallucinations. The segregation of the symptoms into three syndromes is not entirely clear cut: incoherence of speech, which has a high loading on factor 2, also has a moderate loading on factor 3, providing some evidence that it is also associated with delusions and hallucinations.

Psychomotor poverty is the most prevalent syndrome in this group of patients. If definite evidence (i.e. symptom score $\geqslant 2$) for at least one symptom of a syndrome is taken as evidence for the presence of that syndrome, psychomotor poverty is present in 45 cases, reality distortion in 37, and disorganisation in 26. In three cases there is no definite evidence of any of the three syndromes. Thirty-five patients have definite evidence of more than one syndrome, and, of these, 19 exhibit definite evidence of all three syndromes. Scatter plots of factor scores reveal no evidence that the patients segregate into identifiable groups.

The correlations between syndrome scores and age of onset of illness are all near zero. Scores for the psychomotor poverty syndrome are correlated positively with length of illness ($r=0.39$, $P=0.004$), scores for the reality distortion syndrome negatively ($r=-0.36$, $P=0.006$), while scores for the disorganisation syndrome did not correlate with length of illness ($r=0.03$).

There is a trend ($r=-0.26$, $P=0.06$) for scores for the psychomotor poverty syndrome to be associated with lower doses of antipsychotic medication, and there is a significant negative association between scores for psychomotor poverty and use of anticholinergic medication ($r=-0.31$, $P=0.02$). There are no statistically significant associations between severity of Parkinsonism and syndrome scores. The correlations between Parkinsonism and both the disorganisation syndrome and the reality distortion syndrome are virtually zero, while there is a weak correlation between Parkinsonism and scores for psychomotor poverty ($r=0.23$, $P=0.1$).

Discussion

The segregation of chronic schizophrenic symptoms into three syndromes confirmed in this study is partially congruent with Bleuler's (1911) traditional subdivision, insofar as the disorganisation syndrome comprises symptoms characteristic of hebephrenic schizophrenia, and the reality distortion syndrome comprises symptoms characteristic of paranoid schizophrenia. The psychomotor poverty syndrome includes symptoms, such as flattening of affect, which are characteristic of simple schizophrenia, but also includes motor disturbance, suggesting some overlap with catatonic schizophrenia. However, in contrast to the traditional subdivision which sought to identify discrete types of illness, the three syndromes can coexist.

The composition of the syndromes is also partially consistent with positive and negative syndromes. The psychomotor poverty syndrome comprises symptoms generally regarded as negative symptoms, and the reality distortion syndrome comprises symptoms generally regarded as positive symptoms. The emergence of the disorganisation syndrome (which includes certain components of thought disorder) as a syndrome independent of the positive–negative dichotomy indicates a possible resolution of the current controversy in which some authors (Lewine *et al*, 1983) regard disorders of the form of thought as negative symptoms, while others regard them as positive (Crow, 1980).

The identification by Gibbons *et al* (1985) of a syndrome comprising incoherent, irrelevant and wandering speech and inappropriate affect is in accord with the contention that disorders of the form of thought and inappropriate affect constitute a distinct syndrome. However, our finding that incoherence of speech, as defined in the Manchester scale, has a moderate correlation with delusions and hallucinations, suggests that formal thought disorder should not be considered a homogeneous entity. Spohn *et al* (1986) found that "severe forms of thought disorder" responded to neuroleptic treatment, while less dramatic forms of thought disorder were unaffected. While the clinical description of disorders of the form of thought requires further refinement, it is possible that the less bizarre forms of thought disorder identified by Gibbons *et al* (1985), such as irrelevant and wandering speech, are more characteristic of the disorganisation syndrome, while some of the more bizarre forms of thought disorder are more closely related to the reality distortion syndrome.

Bilder *et al* (1985) have also identified a schizophrenic syndrome comprising disorders of the form of thought and bizarre behaviour. This syndrome resembles the disorganisation syndrome, but includes both positive thought disorders and alogia. This difference in detail probably arises from the fact that they used SANS subscale scores as the variables in their factor analysis, whereas this study did not make *a priori* assumptions about the grouping of individual symptoms. The alogia subscale embraces both poverty of speech and poverty of content of speech. In this study, poverty of speech had a high loading in factor 1 (psychomotor poverty) while poverty of content of speech had a high loading in factor 2 (disorganisation).

There is some evidence from other studies that the pattern of symptoms exhibited by schizophrenic patients tends to evolve during the course of the illness. For example, Pfohl & Winokur (1982) found that positive symptoms are prominent in the early stages of the illness, while negative symptoms became more prominent later. In this study, the correlations between syndrome scores and length of illness suggest a tendency for reality distortion symptoms to be more severe early in the illness, and psychomotor poverty symptoms to be more severe late in the illness. This tendency was not found previously (Liddle, 1987*a*), but in that study the range of length of illness was much narrower.

The absence of strong correlations between syndrome scores and dose of antipsychotic medication cannot be taken as evidence that medication does not modify these chronic schizophrenic syndromes, because it is possible that the patient's medication has been adjusted in a way that obscures relationships between medication and symptoms. For example, the observed trend for psychomotor poverty to be associated with lower doses of antipsychotic medication might reflect a tendency for clinicians to prescribe lower doses of medication in patients with marked psychomotor poverty symptoms. The weak positive correlation with severity of Parkinsonism suggests that drug-induced Parkinsonism makes at least a minor contribution to the psychomotor poverty syndrome.

Despite the likelihood that medication modifies the syndromes, the clinical evidence (Bleuler, 1911) suggests that the symptoms comprising these syndromes existed long before antipsychotic medication was used. If medication had differential effects on the different groups of symptoms, this would serve to reinforce the hypothesis that these syndromes differ in their underlying pathophysiological mechanisms.

While medication might induce some confounding effects, it also reduces the potentially confounding influences of acute mental disturbance. An attempt to delineate the persistent pathological processes of schizophrenia by studying patients who could be maintained in a stable state without medication would have the disadvantage that such patients would probably be representative only of those with relatively mild illness.

This study, together with the previous study (Liddle, 1987*a*,*b*), suggests that there are three distinguishable, but related, pathological processes underlying the symptoms of chronic schizophrenic patients. While there is some evidence that medication has differential effects on the three syndromes, it is likely that the syndromes reflect three distinguishable components of the schizophrenic disease process.

References

ANDREASEN, N. C. (1982) Negative symptoms in schizophrenia: definition and reliability. *Archives of General Psychiatry*, 39, 784–788.

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM–III). Washington, DC: APA.

BILDER, R M., MUKHERJEE, S , RIEDER, R. O., *et al* (1985) Symptomatic and neuropsychological components of defect states. *Schizophrenia Bulletin*, 11, 409–419.

BLEULER, E. (1911) *Dementia Praecox or the Group of Schizophrenias* (trans. 1950 by J. Zinken). New York: International Universities Press.

CROW, T. J. (1980) Molecular pathology of schizophrenia. more than one diseases process? *British Medical Journal*, 280, 1–9.

GIBBONS, R. D., LEWINE, R. R J , DAVIS, J. M., *et al* (1985) An empirical test of a Kraepelinian vs. a Bleulerian view of negative symptoms. *Schizophrenia Bulletin*, 11, 390–396.

KRAWIECKA, M., GOLDBERG, D. & VAUGHAN, M. (1977) A standardized psychiatric assessment for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, 55, 299–308.

LEWINE, R. R. J., FOGG, L. & MELTZER, H. Y. (1983) Assessment of negative and positive symptoms in schizophrenia. *Schizophrenia Bulletin*, 9, 368–376.

LIDDLE, P. F. (1987*a*) The symptoms of chronic schizophrenia: a re-examination of the positive–negative dichotomy. *British Journal of Psychiatry*,151, 145–151.

—— (1987*b*) Schizophrenic syndromes, cognitive performance and neurological dysfunction. *Psychological Medicine*, 17, 49–57.

MINDHAM, R. H. S. (1976) Assessment of drug-induced extrapyramidal reactions and the drugs given for their control. *British Journal of Clinical Pharmacology*, 3(suppl 2), 395–400.

PFOHL, B. & WINOKUR, G. (1982) The evolution of symptoms in institutionalized hebephrenic/catatonic schizophrenics. *British Journal of Psychiatry*, 141, 567–572.

SPOHN, H. E., COYNE, L., LARSON, J., *et al* (1986) Episodic and residual thought pathology in chronic schizophrenics. *Schizophrenia Bulletin*, 12, 394–407.

*Peter F. Liddle, BMBCh, BSc, PhD, MRCPsych, *Senior Lecturer in Psychological Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, Ducane Road, London W12 0HS*; Thomas R. E. Barnes, MBBS, MRCPsych, *Senior Lecturer in Psychiatry, Charing Cross and Westminster Medical School, Horton Hospital, Long Grove Road, Epsom, Surrey KT9 8PZ*

*Correspondence

British Journal of Psychiatry (1990), 157, 562–570

Regional Brain Metabolism During Auditory Hallucinations in Chronic Schizophrenia

J. M. CLEGHORN, E. S. GARNETT, C. NAHMIAS, G. M. BROWN, R. D. KAPLAN, H. SZECHTMAN,
B. SZECHTMAN, S. FRANCO, S. W. DERMER and P. COOK

Regions of the brain involved in language and attention were studied using [^{18}F]-fluorodeoxyglucose in PET. In nine chronic DSM–III schizophrenic patients who had persistent auditory hallucinations, ten who had recovered from hallucinations and ten normal controls. In none of the regions examined was metabolic activity significantly different in hallucinating patients compared with that in other groups. However, a pattern of seven significant correlations of metabolic activity between language regions and between frontal and parietal cortex characterised the hallucinating but not the other groups. Three of the seven correlations were significantly greater in hallucinating patients than in the two other groups, and six were greater in hallucinating patients than controls. Metabolism in Broca's region and its right-hemisphere homologue correlated positively and significantly in the hallucinating group, as it did in anterior cingulate and left superior temporal areas, and in right frontal and parietal areas. Hallucination ratings correlated with metabolism in the anterior cingulate region.

This study examines the hypothesis that auditory hallucinations in schizophrenic patients are associated with increased neuronal activity and therefore metabolic activity in the language systems of the brain. This hypothesis is based in part on observations by Penfield & Rasmussen (1950) and Penfield & Perrot (1963) that stimulation of temporal cortex in neurosurgical patients elicited auditory hallucinations – an observation that has been replicated by Mahl *et al* (1964), Horowitz & Adams (1970), and Gloor *et al* (1982). Furthermore, complex partial seizures recorded in the superior temporal region have been associated with auditory hallucinations (Weiser, 1983). Penfield & Rasmussen (1950) observed that audible sounds were elicited by stimulation of right temporal regions as commonly as on the left. However, some observers of brain metabolism in schizophrenia favour the dominant temporal lobe as a location for positive psychotic symptoms (Sheppard *et al*, 1983; Gur *et al*, 1983, 1987).

Furthermore, since auditory hallucinations are accompanied by the mouthing of speech (Gould, 1949; Bick & Kinsbourne, 1987) all components of the language systems of the brain might be involved. Andreasen (1986, 1988) has suggested auditory hallucinations may represent a complex interaction between language regions that generate speech, that encode words, and that put words into a grammatical structure, that is, the temporal lobe language system, Broca's region, and subcortical limbic structures.

Method

Subjects

We examined two groups of chronic schizophrenic patients: ten had auditory hallucinations that had responded to neuroleptics in standard doses, and they no longer reported auditory hallucinations; nine had persistent hallucinations in spite of treatment with neuroleptics at similar doses. All patients were males, and none had concurrent psychiatric or medical diagnoses other than meeting the DSM-III criteria for schizophrenia (American Psychiatric Association, 1980).

Patients were recruited from a rehabilitation programme in which they were monitored day and night. A certain selection bias was thereby introduced as the patients had to appear amenable to rehabilitation to clinicians who selected them for the programme. During the programme they were housed in boarding homes and seen in the rehabilitation centre during the day. Staff in charge of the patients were certain the patients did not take contraband drugs and the patients denied taking them.

Patients had thorough assessments for medical and neurological disorders at the time of their several admissions. Neuropsychological assessment was performed on all patients. All ten of the non-hallucinating patients and six of the nine hallucinating patients had undergone computerised tomography (CT). Ventricular : brain ratios were measured by the method of Golden *et al* (1980).

Patients were assessed neuropsychologically using a battery of 60 tests measuring general intelligence, memory and learning, fluency, problem-solving skills including categorisation, mazes, shifting of set, continuous reaction time, motor speed and dexterity. Most of these measures are in common use in clinical research laboratories and have

---

Presented by J. M. Cleghorn to the 43rd Annual Convention of the Society of Biological Psychiatry, 4–8, May 1988, Montreal, Canada, and, in part, by E. S. Garnett to the Royal College of Psychiatrists, September 1987, London, England.

been used in other studies employing PET in this centre. Thus, in addition to norms for most of the psychological tests available in the literature, there are in-house norms for most of the instruments.

Data from ten normal volunteers previously reported were used as the control values (Szechtman *et al*, 1988). Controls were solicited by advertisement for studies of normal brain function. They were students or hospital employees. All controls were male and physically healthy. They were interviewed with the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) and did not have a history of psychiatric disorder.

Informed consent, following a full explanation of all procedures employed in the study, was obtained from all subjects.

PET procedures

An experienced psychiatric nurse cared for the subjects and inserted a no. 18 angiocatheter into a vein of the forearm. Subjects were injected with 100 MBq [^{18}F]-fluorodeoxyglucose (FDG) while lying on a comfortable stretcher. They were instructed to keep their eyes closed, but were then not spoken to. The only noise was from the air-conditioning system. Arterialised samples of venous blood were taken from the arm not used for [^{18}F] FDG injection for the determination of plasma glucose and [^{18}F] FDG concentrations. Local rates of cerebral glucose metabolism were calculated according to the method of Brownell *et al* (1983).

The Schedule for Affective Disorders and Schizophrenia – Current (SADS–C; Endicott & Spitzer, 1978) was then administered by the nurse so that ratings of positive and negative symptoms, initially obtained on admission, could be checked. The assessment of five negative symptoms (SANS) and four positive symptoms (SAPS) was on six-point scales in which each level of intensity is defined in behavioural terms (Andreasen & Olsen, 1982). The ratings are based on interview data and on behavioural observations by staff and family members.

Patients were asked to report any auditory hallucinations they had during the uptake of [^{18}F] FDG, immediately before the scan, and the hallucination scale of the SAPS was applied to these reports. A rating of three describes the hallucination as vivid, occasional and bothersome; a rating of four, as vivid, frequent, and pervading the patient's life.

Regional distribution of [^{18}F] FDG was measured using the McMaster PET scanner, which has a spatial resolution of 8 mm (full width at half maximum) in the plane and 12 mm in the axial direction (Nahmias, 1984). It can therefore resolve voxels of less than 1 cm^3. At least 10^6 counts were collected, in three minutes, for each slice. The subject's head was held in a perspex restraining device. Alignment in the left–right axis was assured by inspection of a preliminary scan taken at the level of the thalamus.

Typically, 16 slices of the brain (on a plane 5° to the orbitomeatal line) were obtained sequentially from the level of the hippocampal gyri up to the level of cortical vertex. Although each slice was 15 mm thick, it overlapped its predecessor by 10 mm. Using this procedure, the slice with greatest volume of grey matter of interest for each subject can be identified and selected. For this study, three slices were selected for analysis (Fig. 1). The *lowest slice* (midthalamic–midstriatal slice) contained the largest amount of thalamic and striatal tissue from the temporal cortex; auditory cortex is defined as segments 10 and 11 (Fig. 2). The procedure for dividing the cortex into segments is described below. The *middle slice* (mid-callosal slice) passed above the basal ganglia through the midportion of the dorsoventral extent of the corpus callosum.

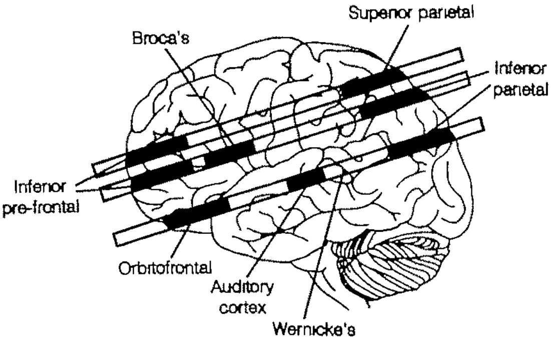


FIG. 1 The three slice levels for which data are reported. The dark areas indicate the segments of tomographic slices analysed.

Broca's region was located on the basis of maps of stimulation that produced speech arrest. These maps were photographed at neurosurgery by Penfield & Roberts (1959)

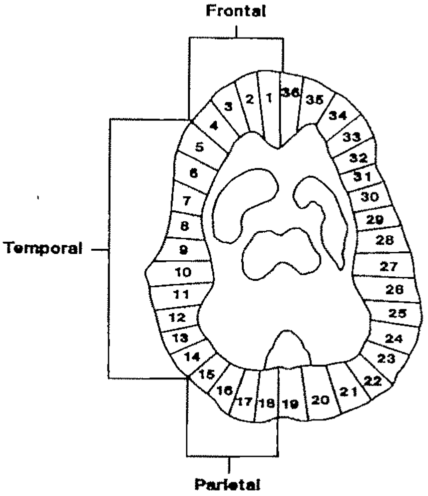


FIG. 2 The cortical rim is divided into 36 segments each spanning 10°. The borders were defined by an edge-detecting algorithm Central structures are corpus striatum and, posteriorly, thalamus This is the low slice in Fig. 1. In the middle slice no structures are visible in the centre of the brain. Broca's region was defined to include segments 6, 7 and 8 in that slice (see text) In the high slice the cingulate gyrus is visible as well as the rim of the cortex.

and subsequently confirmed and elaborated by Ojemann (1983). Broca's region was identified by measuring the distance from the anterior pole to the anterior border of Broca's on the C–M line and similarly for the posterior border to the posterior pole on the Penfield map. These measurements were transformed to proportions and imposed on the PET slice. The slice was found by similar measurements to correspond to a region which approximately bisected Broca's. Segments 5 and 9 are at the anterior and posterior borders of Broca's. To be certain not to use data from areas outside Broca's region, we utilised metabolic data from segments, 6, 7 and 8 in the mid-slice (Fig. 1), which is higher than the slice in Fig. 2. This definition of Broca's region appears to correspond with the definition used by Horwitz *et al* (1984) in their PET studies.

Wernicke's area is defined as segments 9–13 in the lower slice, the posterior half of the superior temporal gyrus. The posterior superior component of Wernicke's area was not sampled.

Auditory cortex is defined as segments 10 and 11 on the left side of the low slice and its right-hemisphere homologue as segments 26 and 27 on the right (Figs 1, 2).

Finally, the ***highest slice*** (supracallosal slice) cut just above the vertex of the corpus callosum and passed through the cingulate cortex. The cingulate region is defined as the strip of anterior–posterior cortex immediately above the corpus callosum. It was easily identified in all but one subject. It was cut in half to represent approximately those portions of anterior and posterior cingulate that lie inside a single tomographic slice. The anterior and posterior borders are taken to be 25% of the distance from the anterior and posterior poles of the slice.

Identification of these three levels was consistent from subject to subject because of the large number of slices of each brain taken. We have presented pictures of the three slices elsewhere (Szechtman *et al*, 1988).

To examine the regional distribution of glucose metabolism in the cortex, a computer algorithm was developed to divide the cortical rim of each hemisphere of a slice into 18 regions, as follows. To delineate the cortical rim, the image was preprocessed with a local-intensity subrange filter. A unit-width connected edge was then extracted using an automatic contour-following algorithm. The threshold value was set to 60% of the maximum pixel value in the image. In portions of the image where automatic extraction was inappropriate (e.g. because it would include deep grey-matter structures) the operator traced the most probable contour. The centre of mass of the outer contour was then computed and each hemisphere divided into 18 wedges by tracing radial lines from this centre every 10°. The area of the wedge was governed by the thickness of the cortical rim and the angle between two consecutive radial lines. This, together with the slice thickness, defined a volume of cortex. The mean glucose metabolism in each of these volumes was obtained. The division of the cortical rim is shown in Fig. 2: the most anterior region on the left is numbered 1 and the most posterior region is 18 (bilaterally). On the right the most anterior region is 36 and the posterior pole is 19.

The corpus striatum and thalamus were identified using the same edge-detection and feature-extraction algorithms (Fig. 2).

Results

Matching of subject groups

The two patient groups were comparable in age, education, neuroleptic use, age of onset, years of illness, and number of episodes (Table I). Intelligence was in the normal range. One patient had involuntary movements of the trunk of the body which had been present before treatment. None of the other patients showed involuntary movements. Ventricular : brain ratios (Table II) were not significantly different from each other in the 16 patients undergoing CT. However, in spite of careful matching, it was found that six of the nine patients with persistent auditory hallucinations had a history of pronounced and enduring (2–12 years) drug abuse such that they met DSM-III criteria for substance abuse in the past.

Table II also displays the mean symptom scores of the 19 patients. There were no significant differences between the two groups with respect to symptom scores, other than hallucinations.

An analysis of variance on the 60 neuropsychological tests revealed no significant differences between patient groups. However, schizophrenic patients had a mean full-scale IQ of 100 (s.d. 15) compared with 122.7 for controls (s.d. 6.2). This difference precludes comparisons of IQ between these groups. Covariance analysis is not an adequate substitute for correct matching (Adams *et al*, 1984). Other demographic data for controls are: mean age 27.6, s.d. 4.8 years (range 22–38), mean level of education 17.8, s.d. 2.8 years (range 13–23), and all controls but one were right handed.

Regional brain $[^{18}F]$ FDG metabolism

All nine hallucinators reported hallucinations at the end of $[^{18}F]$ FDG uptake. Six reported single repetitive voices and three reported hearing conversations between two voices (see Table II for hallucination scores).

There were no significant differences by ANOVA in mean (s.d.) glucose metabolic rate between groups (low, middle, high slices respectively: 36 (26), 37 (25), 40 (25), hallucinators; 41 (15), 41 (15), 40 (15), non-hallucinators).

Metabolism in each region of interest (Fig. 1) relative to the mean for the slice that included it was compared by a two-way analysis of variance for each region. There were no significant differences between the groups in any of the frontal and/or parietal regions. No laterality effect was found. However, in the lowest slice, a group-by-laterality interaction was observed ($F(1,17)=4.47$; $P=0.049$). Examination of the ratios (activity in region : mean for slice) revealed that the orbitofrontal region in the hallucinating patients on the left side (0.990, s.d. 0.060) was more active than that on the right (0.988, s.d. 0.70), and the opposite was the case in the non-hallucinating group (1.007, s.d. 0.096, left, v. 1.011, s.d. 0.86, right).

The frontal : parietal activity ratios were also examined at each slice level. No group differences nor laterality differences, nor group-by-laterality interactions were detected. Next, metabolism in Broca's region in the frontal cortex was examined. The groups did not differ in either ratios

Demographic and medical data

Identification no.	*Age: years*	*Handedness*	*No. of years in education*	*No. of years on medication*	*Age at onset: years*	*Years of illness*	*No. of episodes*	*Current medication*
Hallucinating (n = *9)*								
501[1]	27	Right	13	4	22	5	1	Pipotiazine palmitate, 100 mg monthly
502[1]	30	Right	10	9	12	18	4	Haloperidol, 10 mg daily; Flupenthixol decanoate, 60 mg every 2 weeks
503[1]	31	Left	15	7	23	7	4	Haloperidol, 150 mg every 4 weeks
505[1]	23	Right	13	4	18	4	1	Perphenazine, 40 mg daily
506[1]	31	Right	13	6	21	9	6	Flupenthixol decanoate, 100 mg every 2 weeks
801[1]	41	Right	7	11	30	11	6	Haloperidol, 20 mg daily
802	28	Mixed	13	7	21	7	4	Trifluoperazine, 30 mg daily
803	27	Right	13	5	21	7	7	Haloperidol, 25 mg every 3 weeks
804	27	Right	10	6	19	8	5	Haloperidol, 200 mg every 2 weeks
Means	29		12	6	21	8	4	
s.d.	5		2	2	5	4	2	
Non-hallucinating (n = *10)*								
601[1]	32	Right	13	12	20	12	5	Pipotiazine palmitate, 37.5 mg every 2 weeks
602[1]	28	Right	10	8	20	8	3	Perphenazine, 24 mg daily
603[1]	22	Right	13	4	18	4	8	Perphenazine, 24 mg daily
604	19	Mixed	12	2	17	2	1	Flupenthixol decanoate, 40 mg every 3 weeks
605[1]	25	Right	14	5	20	5	2	Fluphenazine decanoate, 12.5 mg every 3 weeks
606[1]	34	Mixed	9	14	20	14	5	Fluphenazine decanoate, 50 mg every 2 weeks
702	23	Right	10	–[2]	19	5	2	Tricyclic dibenzoxazepine, 40 mg daily
703	23	Right	10	–[2]	15	7	12	Flupenthixol decanoate, 200 mg every 2 weeks
704	33	Mixed	9	–[2]	18	14	4	Fluphenazine decanoate, 25 mg every 2 weeks, intramuscularly
705	33	Right	8	16	16	16	3	Haloperidol, 5 mg daily
Means	27		11	9	18	9	5	
s.d.	5		2	5	2	5	3	

1. Data on 11 of these patients were presented in a report of neuroleptic effects on cerebral metabolism, and the same identification numbers are used here as in that study (Szechtman *et al*, 1988)
2. Data not available.

for left or right areas, or the mean values for right and left, by analysis of variance. (Full data are available from author, on request.) A similar analysis was performed for auditory cortex, and no significant differences between the groups were found by analysis of variance for ratios of activity on the left or right, or that for the mean of left and right. When metabolism in the entire superior temporal region was examined, there were no significant differences between the patient groups.

In the posterior temporal region representing part of Wernicke's region, an asymmetry was found. Analysis of variance revealed a laterality effect ($F(1,17)=5.35$, $P=0.033$) such that the relative metabolism in this region is greater on the right side (1.071, s.d. 0.073, for hallucinators, 1.055, s.d. 0.070, for non-hallucinators) than on the left (1.030, s.d. 0.058; 1.010, s.d. 0.064, respectively) in both groups.

No such asymmetry was found in the middle slice. Metabolism in the striatum (caudate and putamen) and in the thalamus was also examined. No significant differences between patient groups were detected.

The ratios of metabolic activity in anterior and posterior cingulate regions to the mean for the slice showed no significant differences between patient groups (mean for hallucinators, anterior 0.90, s.d 0.07, posterior 0.90, s.d. 0.07; mean for non-hallucinators, anterior 0.89, s.d. 0.07, posterior 0.88, s.d. 0.09). There were only eight hallucinating subjects in this analysis because the cingulate could not be imaged well in one subject.

We previously reported a comparison of 11 of the schizophrenic subjects and the 10 controls (Szechtman *et al*, 1988). The same ID numbers are used in this report. The addition of eight new subjects in the present study did not alter the earlier results. The reanalysis including the new subjects is as follows. The midparietal region is significantly less active in schizophrenics than in controls ($F(1,27)=6.49$, $P=0.017$). The high pre-frontal region is significantly more active in patients than controls ($F=1,27)=4.72$, $P=0.039$). The superior temporal region (segments 5–14 and 23–32) in the low slice is significantly more active in schizophrenics than in controls ($F(1,27)=6.94$, $P=0.014$). In addition, a supramarginal region (segments 9–13) in the middle slice is significantly more active in patients than controls ($F(1,27)7.82$, $P=0.004$).

Data on the cingulate gyrus were not reported previously (Szechtman *et al*, 1988). There are no significant differences between the 19 patients and the 10 controls for either anterior or posterior cingulate.

Correlations

Pearson's correlations were computed on the regions involved in language (temporal, auditory and Broca's) and attention (frontal and parietal) (Table III). The language regions right and left comprise six variables, and frontal and parietal at three slice levels, right and left, 12 variables.

A pattern of significant correlations between regions was observed in the hallucinating patients and not in the non-hallucinating patients, nor in normal controls previously reported (Table III) (Fig. 3). There were significant and positive correlations for metabolic activity in Broca's region and that in its right-hemisphere homologous area, and for that region and the right superior temporal region. A significant negative correlation between the right hemisphere region homologous for Broca's and the left superior temporal cortex was obtained. Thus, metabolic activity covaries in certain language-related areas in hallucinating patients but not in patients whose hallucinations had responded to neuroleptics nor in normal controls. However, auditory and Wernicke's cortex metabolism, both defined as part of the superior temporal slice, did not correlate significantly with any region in any of the groups.

In addition, there were significant negative correlations between metabolism in the right pre-frontal parietal high and mid-slice regions in the hallucinating group, and between left high frontal and parietal regions.

There were no significant correlations between Broca's regions and other frontal or parietal regions. In the controls, there was a correlation between right orbitofrontal and inferior parietal regions.

Those correlations which significantly ($P<0.01$) differentiated the groups were then further examined by Fisher's test (Hays, 1975) (Table III). The hallucinating patient group had significantly higher correlations than the non-hallucinating group between the following: high right frontal and parietal regions, right and left Broca's regions, and anterior cingulate and left superior temporal.

When the hallucinating patients and controls were compared, the following regions in addition to those above were significantly different ($P<0.01$): right midfrontal and parietal, left high frontal and parietal, left and right temporal and right Broca's.

Correlations were also calculated between auditory hallucination scores, measured immediately before the scan, and metabolism in the various regions. The hallucination scores ranged from two to five and correlated positively and significantly with anterior cingulate metabolism ($r=0.82$, $P<0.01$, $n=8$). There were non-significant trends in left-sided regions in auditory cortex, and in Broca's cortex.

Discussion

This study, the first to our knowledge to compare hallucinating and non-hallucinating patients with PET, failed to find patterns of relative regional brain metabolism that differentiated the patient groups. A larger sample would be required to state with certainty that no such difference exists. It is also possible that no difference can be detected because the signals emitted during intermittent hallucinatory episodes were not sufficiently strong and enduring to be reflected in regional metabolic changes.

The group-by-laterality interaction in the orbitofrontal region barely reached statistical significance and will not be interpreted since it is one of numerous comparisons and had not been predicted. Not all brain regions were examined and it is possible that differences may be found in other regions such as hippocampus and amygdala through which access to verbal (and other) memories is made.

TABLE II
Ventricular:brain ratios and symptoms during [^{18}F] FDG uptake

Identification no.	*Ventricular : brain ratio (%)*[1]	*Symptoms*								
		Affective blunting	*Alogia*	*Avolition/ apathy*	*Anhedonia*	*Attention*	*Hallucinations*	*Delusions*	*Bizarre behaviour*	*Thought disorder*
Hallucinating (n = 9)										
501	5.2	1	0	1	1	0	3	0	0	0
502	4.0	2	1	1	3	1	3	1	0	0
503	3.5	3	0	3	5	2	4	0	0	0
505	3.7	0	0	0	1	1	4	0	0	0
506	6.5	0	0	2	3	0	5	4	0	0
801	7.5	1	0	3	4	2	2	1	0	0
802	–[2]	2	1	0	3	0	3	2	0	0
803	–[2]	3	0	4	3	0	3	2	0	0
804	–[2]	1	0	2	4	0	3	4	4	0
Means	5.1	1.4	0.2	1.8	3.0	0.7	3.3	1.6	0.4	0
s.d.	2.4	1.3	0.4	1.4	1.3	0.9	0.9	1.6	1.3	0
Non-hallucinating (n = 10)										
601	7.8	0	0	0	0	1	0	0	0	0
602	8.3	1	0	0	3	1	0	0	0	0
603	4.5	2	0	0	2	0	0	2	1	0
604	4.1	1	0	0	3	0	0	0	0	1
605	10.7	0	1	0	1	1	0	0	0	0
606	4.4	0	0	1	0	0	0	0	0	0
702	4.2	3	2	1	4	2	0	2	1	0
703	10.5	0	0	0	0	0	0	0	0	0
704	5.7	0	0	0	1	1	0	0	0	0
705	7.5	0	0	0	3	3	0	0	0	0
Mean	6.8	0.7	0.3	0.2	1.7	0.9	0	0.4	0.2	0.1
s.d.	2.6	1.1	0.7	0.4	1.5	1.0	0	0.8	0.4	0.3

1. At maximum ventricular width.
2. Data not available in chart.

We have defined language regions of the brain in terms of data from brain-stimulation maps (Penfield & Roberts, 1959; Ojemann, 1983). In fact PET data on word production and processing suggests other regions may also be involved (Petersen *et al*, 1988). In future, it will be possible to add precision to localisation by identifying each individual patient's response to verbal and listening tasks.

Normal subjects do show higher rates of glucose consumption in Broca's and left temporal areas (Mazziotta *et al*, 1982; Damasio *et al*, 1986) when speaking or listening. However, our hallucinating schizophrenic patients did not show such a pattern of metabolic activation. This failure of cortical glucose activation may mean that these patients do not attend to their voices, as is often the case with chronic schizophrenics (Hamilton, 1984). Such a suggestion is consistent with the observations of Hubel & Wiesel (1959) that neurones in the auditory cortex of experimental animals respond to sounds only when the animal appears to be paying attention to them. Furthermore, habituation is the cortical response to repetitive stimuli and auditory hallucinations were repetitive in this study. This may explain the absence of regional metabolic differences between our patient groups.

We previously reported clear differences between 11 of our patients and the same control group (Szechtman *et al*, 1988). Patients were relatively hyperfrontal, hypertemporal and hypoparietal when compared with controls. The addition of eight patients in the present report did not alter this pattern. The fact that auditory hallucinations do not influence this pattern argues against their salience for our cortical metabolic map of schizophrenia which has now been replicated in a sample of eight drug-naive patients (Cleghorn *et al*, 1989).

TABLE III
Correlations of metabolic activity in brain regions involved in language and attention

Region	*Hallucinating patients (n = 9)*	*Non-hallucinating patients (n = 10)*	*Controls (n = 10)*
Right frontal and parietal [1]			
high slice[1]	−0.94**	−0.47	−0.14
middle slice	−0.90**	−0.57	−0.07
low	−10.46	−0.64	−0.89**
Left frontal and parietal			
high slice	−0.85*	−0.55	−0.10
middle slice	−0.32	−0.60	−0.39
low slice	−0.37	−0.65	−0.21
Right temporal and:			
right Broca's	+0.87*	+0.50	+0.18
left Broca's	+0.54	−0.41	−0.07
Left temporal and:			
right Broca's	−0.81*	−0.48	+0.34
left Broca's	+0.32	+0.30	+0.42
Right and left Broca's[1]	+0.78*	−0.07	+0.38
Anterior cingulate and:			
left superior temporal[1]	+0.93**	−0.65	+0.62
Broca's	−0.76	+0.09	+0.16

1. Regions in which the correlations were significantly greater in hallucinating v. non-hallucinating groups by Fisher's test.
*$P<0.01$, **$P<0.001$.

In both patient groups metabolic activity was greater on the right than on the left in the posterior temporal or Wernicke's region (in Fig. 2, regions 11–14 left and 24–27 right). The same pattern has been observed in normal subjects (Szechtman *et al*, 1988). This pattern may simply reflect anatomical asymmetry in the temporal lobe, described by Geschwind & Levitsky (1968).

Although no single brain region involved in speech and language differentiated the two groups, a pattern of correlations between regions distinguished hallucinating from non-hallucinating schizophrenic patients. The pattern of correlations implicates regions related or homologous to language functions. Left and right Broca's, anterior cingulate and left superior temporal cortex covary positively. Anterior cingulate metabolism is the only region found to correlate positively and significantly with hallucination scores.

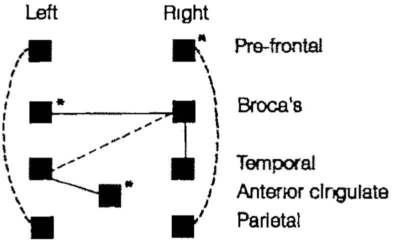


FIG 3 A pattern of significant ($P<0.01$) correlations (—— negative, —— positive) between regions of brain metabolism found only in patients with auditory hallucinations, not in those whose hallucinations had long ago disappeared with treatment, nor in normal controls (see Table III for values). Asterisks indicate correlations that were significantly greater in the hallucinating than non-hallucinating patients.

The anterior cingulate area is part of an anterior attentional system engaged in the selection for action from auditory perception of words with different meanings (Petersen *et al*, 1988). The region has direct anatomical and functional links with superior-temporal cortex concerned with species-specific phonation (Müller-Preuss *et al*, 1980) and is used for discriminating intended vocalisations from other auditory stimuli. McKenna (1987) has speculated that this connection might be deranged in an auditory hallucination, which is not an intended vocalisation (Hoffman, 1986). According to MacLean (1985) the anterior cingulate area is required for normal attachment behaviour and play. Ablation of the region eliminates spontaneous speech and affective expression. Electrical stimulation heightens attention and positive affect and produces impatient leg movements, like akathisia.

Our data suggest that the cingulate and superior temporal regions are involved in auditory hallucination but we do not have evidence that they are deranged, However, Benes *et al* (1987) have reported histopathological evidence of an elevated proportion of vertical axons in the cingulate cortex of schizophrenics.

It is uncertain whether these particular neurones are connected to the superior temporal region.

Studies of normal subjects' cerebral blood flow have revealed that blood flow in highly specialised regions such as auditory and Broca's cortex have low correlations with that in other frontal regions, while their less specialised counterparts on the right side are normally coupled with frontal regions. Homologous regions in each hemisphere tend to be negatively correlated with regard to blood flow when one unilateral region is engaged in its specialised function (Prohovnik *et al*, 1980). None of these patterns of correlation in normal subjects was observed in our hallucinating patients, who are engaged in the specialised task of articulating and hearing auditory hallucinations.

The significance of correlations between metabolic values in different regions of the brain is not well understood. There is some evidence that correlation of brain metabolism in one region with another may reflect actual pathology. Patients with Alzheimer's disease have been found to have significantly fewer reliable partial-correlation coefficients for activity in frontal and temporal–parietal lobe regions, an observation consistent with the location of pathology in Alzheimer's disease (Horwitz *et al*, 1987). A meaningful pattern of correlations of regional glucose metabolism has been found in autism (Horwitz *et al*, 1988). Studies of the functional coupling of brain regions in the awake rat by audioradiography suggest that the correlational technique demonstrates functional interactions among brain areas and provides a method to explore anatomical and functional relationships (Soncrant *et al*, 1986).

Two studies have reported correlations of many brain regions in schizophrenics and controls (Clark *et al*, 1985; Volkow *et al*, 1988). Both studies reported fewer significant correlations in schizophrenics than in normals. Our data are not comparable since most correlations examined involved language regions.

The history of severe substance abuse elicited from six of nine hallucinating patients deserves comment since it challenges the generalisability of our findings. The effect of prior drug abuse on the onset and course of schizophrenia is imperfectly understood (Bowers, 1987). Half of such patients have a poor outcome at five years and in this respect are similar to many schizophrenics. Our drug-abusing schizophrenic patients fit this group. Many other drug-abusing psychotic patients have a good pre-morbid history and are diagnosed as atypical psychotics (Bowers, 1977). Our patients do not share those characteristics. While we observed no differences in brain metabolism related to a history of drug abuse, studies comparing brain metabolism and chemistry in schizophrenics with and without a history of substance abuse are warranted.

Patients in this study were all receiving standard doses of neuroleptic drugs. Little effect of these drugs on cerebral cortex was detected when we compared relative regional cortical metabolism in 11 of the patients reported here (Table I) with drug-naive patients (Szechtman *et al*, 1988). In the striatum, however, neuroleptic-treated patients had significantly greater relative metabolism than controls, but hallucinating and non-hallucinating patients, all medicated, did not differ from each other.

In this study, auditory hallucinations were associated with a pattern of high correlations between some but not all brain regions related to language and non-verbal vocal expression. It will be important in future to determine experimentally whether a similar pattern of correlations is associated with normal speaking and listening. In addition, the pattern of cortical relations should be compared when patients are and are not hallucinating.

If schizophrenic patients show the same pattern when listening to spoken words as when hallucinating, and if this pattern differentiates them from non-hallucinating patients and normal controls, an anomaly in the location of verbal processing could be identified. On the other hand, if the cortical pattern of listening schizophrenics and normals does not differ, but the pattern we described occurs only during auditory hallucinations, then it could be inferred that a circuit not normally used for language processing is activated during auditory hallucinations. A method for distinguishing between these possibilities has been described by Posner *et al* (1988).

Acknowledgement

This study was supported in part by grants from the MRC of Canada (MA 10214) and the NIMH (ROI MH 44073).

References

Adams, K. M., Brown, G. G. & Grant, I. (1984) Analysis of covariance as a remedy for demographic mismatch of research subject groups: some sobering simulations. *Journal of Clinical and Experimental Neuropsychology*, 7, 445–462.

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM–III). Washington, DC: APA.

Andreasen, N. C. (ed) 1986) *Can Schizophrenia be Localized in the Brain?* Washington, DC: APA.

—— (1988) Brain imaging: applications in psychiatry. *Science*, 239, 1381–1388.

—— & Olsen, S. (1982) Negative v. positive schizophrenia: definition and validation. *Archives of General Psychiatry*, 39, 784–788

Benes, F. M., Majocha, R., Bird, E. D., *et al* (1987) Increased vertical axon counts in cingulate cortex of schizophrenics. *Archives of General Psychiatry*, 44, 1011–1021

Bick, P A. & Kinsbourne, M. (1987) Auditory hallucinations and subvocal speech in schizophrenic patients. *American Journal of Psychiatry*, 144, 222–225.

Bowers, M. B. (1977) Psychoses precipitated by psychotomimetic drugs: a follow-up study. *Archives of General Psychiatry*. 34, 832–835.

—— (1987) The role of drugs in the production of schizophreniform psychoses and related disorders. In *Psychopharmacology: The Third Generation of Progress* (ed. H. Meltzer) New York: Raven Press.

Brownell, G L., Kearfott, K. J., Kairento, A-L, *et al* (1983) Quantitation of regional cerebral glucose metabolism. *Journal of Computer Assisted Tomography*, 7, 919–924.

Clark, C., Carson, R., Kessler, R., *et al* (1985) Alternative statistical models for the examination of clinical positron emission tomography/fluorodeoxyglucose data. *Journal of Cerebral Blood Flow and Metabolism*, **5**, 142–150.

Cleghorn, J. M., Garnett, E. S., Nahmias, C . *et al* (1989) Increased frontal and reduced parietal glucose metabolism in acute untreated schizophrenia. *Psychiatry Research*, **28**, 119–133

Damasio, A., Bellugi, V , Damasio, H., *et al* (1986) Sign language aphasia during left hemisphere amytal injection. *Nature*, **322**, 363–365.

Endicott, J. & Spitzer, R. L. (1978) A diagnostic interview: the Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, **35**, 837–84.

Geschwind, N. & Levitsky, W (1968) Human brain – left and right asymmetries in temporal speech region. *Science*, **161**, 186–187.

Gloor, P , Olivier, A., Quesney, L. F., *et al* (1982) The role of the limbic system in experimental phenomena of temporal lobe epilepsy. *Annals of Neurology*, **12**, 129–144.

Golden, C., Moses, J , Zelazowski, R., *et al* (1980) Cerebral ventricular size and neuropsychological impairment in chronic schizophrenics. *Archives of General Psychiatry*, **37**, 619–626.

Gould, L. N. (1949) Auditory hallucinations in subvocal speech: objective study in a case of schizophrenia. *Journal of Nervous and Mental Disease*, **109**, 418–427.

Gur, R. E., Skolnick, B. E., Gur, R. C., *et al* (1983) Brain function in psychiatric disorders: I. Regional cerebral blood flow in unmedicated schizophrenics. *Archives of General Psychiatry*, **40**, 1250–1254.

——, Resnick, S. M., Alavi, A., *et al* (1987) Regional brain function in schizophrenia. I. A positron emission tomography study *Archives of General Psychiatry*, **44**, 119–125.

Hamilton, M. (1984) *Fish's Schizophrenia* New York: Wright.

Hays, W. M (1975) *Statistics: Probability, Inferences and Decision*. New York: Holt, Rinehart & Winston.

Hoffman, R. E. (1986) Verbal hallucinations and language production processes in schizophrenia. *Behavioral and Brain Sciences*, 9, 503–548.

Horowitz, M. J. & Adams, J. E. (1970) Hallucinations on brain stimulation: evidence for revision of the Penfield hypothesis. In *Origin and Mechanism of Hallucinations* (ed. W. Keup) New York: Plenum.

Horwitz, B., Duara, R. & Rapaport, S. I. (1984) Intercorrelations of glucose metabolic rates between regions: application to healthy males in a state of reduced sensory input. *Journal of Cerebral Blood Flow and Metabolism*, 4, 484–499.

——, Grady, C. L., Schlageter, N. L., *et al* (1987) Intercorrelations of regional cerebral glucose metabolic rates in Alzheimer's disease *Brain Research*, **407**, 294–306.

——, Rumsey, J. M., Grady, C. L., *et al* (1988) The cerebral metabolic landscape in autism. *Archives of Neurology*, **45**, 749–755.

Hubel, D. H. & Wiesel, T. N. (1959) Receptive fields of single neurones in the cat's striate cortex. *Journal of Physiology*, 574–591.

MacLean, P. D. (1985) Brain evolution relating to family play and the separation call. *Archives of General Psychiatry*, **42**, 405–417

Mahl, G. F., Rothenberg, A., Delgado, J. M. R., *et al* (1964) Psychological response in the human to intracerebral electrical stimulation. *Psychosomatic Medicine*, **26**, 337–368.

Mazziotta, J. C., Phelps, M. E., Carson, R. E., *et al* (1982) Tomographic mapping of human cerebral metabolism: sensory deprivation. *Annals of Neurology*, **12**, 435–444.

McKenna, P J. (1987) Pathology, phenomenology and the dopamine hypothesis of schizophrenia. *British Journal of Psychiatry*, **151**, 288–301.

Muller-Preuss, P., Newman, J. D. & Jurgens, U (1980) Anatomical and physiological evidence for a relationship between the 'cingular' vocalization area and the auditory cortex in the squirrel monkey. *Brain Research*, **202**, 307–315.

Nahmias, C. (1984) The McMaster positron emission tomograph. design and evaluation. *Nuclear Instruments and Methods*, 1, 113–114.

Ojemann, G. A (1983) Organization for language from the perspective of electrical stimulation mapping. *Behavioral and Brain Sciences*, 6, 189–230.

Penfield, W., Rasmussen, T. (1950) *The Cerebral Cortex of Man*. New York: Macmillan.

—— & Roberts, L. (1959) *Speech and Brain Mechanisms*. New Jersey: Princeton University Press.

—— & Perrot, P. (1963) The brain record of auditory and visual experience. *Brain*, **86**, 595–596.

Petersen, S E., Fox, E. T., Posner, M. I., *et al* (1988) Positron emission tomography studies of the cortical anatomy of single word processing. *Nature*, 331, 585–589.

Posner, M. I , Petersen, S E., Fox, P T., *et al* (1988) Localization of cognitive operations in the human brain. *Science*, **240**, 1627–1631.

Prohovnik, I., Hakansson, K. & Risberg, J. (1980) Observations on the functional significance of regional cerebral blood flow in "resting" normal subjects. *Neuropsychologia*, **18**, 203–217

Sheppard, G., Gruzelier, J., Manchanda, R., *et al* (1983) ^{15}O positron emission tomographic scanning in predominantly never-treated acute schizophrenic patients. *Lancet*, *ii*, 1448–1452.

Soncrant, T. T., Horwitz, B., Holloway, H. W., *et al* (1986) The pattern of functional coupling of brain regions in the awake rat. *Brain Research*, **369**, 1–11.

Szechtman, H., Nahmias, C., Garnett, E. S., *et al* (1988) Effect of neuroleptics on altered cerebral glucose metabolism in schizophrenia. *Archives of General Psychiatry*, **45**, 523–532.

Volkow, N. D., Wolf, A. P., Brodie, J. D., *et al* (1988) Brain interactions in chronic schizophrenics under resting and activation conditions *Schizophrenia Research*, 1, 47–53.

Wieser, H. G (1983) Depth recorded limbic seizures and psychopathology. *Neuroscience and Biobehavioural Review*, 7, 427–440.

*J. M. Cleghorn, MD, *Professor of Psychiatry, McMaster University, 1200 Main Street West, Hamilton, Ontario Canada L8N 3Z5*; E. S. Garnett, MB, *Professor of Radiology, Director, Nuclear Medicine Department, McMaster University*; C. Nahmias, *Associate Professor, Radiology, McMaster University*; G. M. Brown, MD, PhD, *Professor, Biomedical Sciences and Psychiatry, McMaster University*; R. D. Kaplan, PhD, *Associate Professor, Psychiatry, McMaster University*; H. Szechtman, PhD, *Associate Professor, Biomedical Sciences, McMaster University*; B. Szechtman, BA, *Research Assistant, Psychiatry, McMaster University*; S. Franco, RN, *Research Nurse, Psychiatry, McMaster University*; S. W. Dermer, MD, *formerly Executive Director, Hamilton Program for Schizophrenia, Associate Professor of Psychiatry, McMaster University*; P. Cook, MD, *Executive Director, Hamilton Program for Schizophrenia, Assistant Professor of Psychiatry, McMaster University*

*Correspondence

British Journal of Psychiatry (1990), 157, 571–577

A Trial of Family Therapy versus a Relatives' Group for Schizophrenia
Two-Year Follow-up

J. LEFF, R. BERKOWITZ, N. SHAVIT, A. STRACHAN, I. GLASS and C. VAUGHN

The results are reported of a two-year follow-up of a trial of family sessions in the home (including patients) (12 families) versus a relatives' group (excluding patients) (11 families). Subjects were patients with schizophrenia living in high face-to-face contact with high-EE relatives. Patients were maintained on neuroleptic drugs for two years where possible. Relatives' critical comments and hostility were significantly lowered by nine months, but no significant changes occurred subsequently. Relatives' overinvolvement reduced steadily throughout the trial, and reduction in relatives' EE, either alone or in combination with reduced face-to-face contact, appeared to be associated with a lower relapse rate. The relapse rates for patients in the family-therapy and relatives'-group streams were 33% and 36% at two years. When these data were combined with the results of a previous trial, it was found that patients in families assigned to any form of social intervention had a two-year relapse rate of 40%, significantly lower than the 75% relapse rate for patients whose families were offered no help. We therefore recommend that relatives' groups are established in conjunction with some family sessions in the home for patients at high risk of relapse.

Over the last decade, seven trials have been conducted to evaluate the effectiveness of family treatment in combination with maintenance neuroleptic drugs in preventing relapse of schizophrenia. Only one of these failed to demonstrate a significant effect for family treatment, and this trial, in contrast to the others, utilised psychodynamic group treatment for family members (Dulz & Hand, 1986). All the others (Goldstein *et al*, 1978; Leff *et al*, 1982; 1989; Falloon *et al*, 1982; Hogarty *et al*, 1986; Tarrier *et al*, 1988) employed similar combinations of educational, problem-solving and structural approaches (Leff, 1985; Strachan, 1986). The consistent findings of this body of research indicate the value of including family interventions alongside drug treatment in the management of schizophrenia.

The question has moved on from whether family interventions are worthwhile to how they may best be delivered. In our first trial (Leff *et al*, 1985) we found that a package of interventions (a short education programme, a relatives' group, and family sessions in the home) was significantly more effective in reducing the relapse rate of schizophrenia than no help offered to the control group of relatives. Family sessions in the home were offered to relatives who were unwilling or unable to attend the relatives' group. A better clinical outcome for the patients was evident at nine months and at two years. In our second trial (Leff *et al*, 1989) the intention was to separate the elements of the package and compare their relative effectiveness. We did not consider that the education programme merited separate evaluation as it was short (two sessions) and unlikely by itself to effect substantial changes in the family. This assumption was later supported by Tarrier *et al* (1988), who found an 'education-only' condition to be no more effective in reducing relapse than no help offered to relatives. Consequently our trial involved the comparison of education plus a relatives' group with education plus family therapy in the home. In employing this design we were not only evaluating the comparative effectiveness of the two forms of intervention, but also addressing the question of whether desired changes in the family could be achieved equally well without involving the patient (relatives' group) as with the patient's active participation (family therapy).

The disadvantage of this design is its rigidity compared with the flexibility of the original package of interventions. Thus in the second trial, if relatives were assigned to the relatives' group, but did not attend, we were constrained by the design not to offer them family sessions. In the event, out of the 11 families randomly assigned to the relatives' group, five failed to attend even once. By contrast, the effort made by the therapists to visit families at home was greatly appreciated by them, and in only one of the 12 families was there any resistance to the sessions. Although this difference in uptake hindered comparisons, it threw light on the relationship between non-compliance by relatives and the outcome for patients. Patients living with relatives who failed to attend the group had the highest relapse rate over nine months of any subgroup of patients in the two trials (Leff *et al*, 1989).

Here we present the findings of a two-year follow-up of the second trial. The interest lies in whether the benefits of intervention evident at nine months persist until two years, despite a considerably reduced therapeutic input.

Method

The details of the method are given by Leff *et al* (1989). In brief, patients were included if they were assigned to a schizophrenic class by the CATEGO program on the basis of the Present State Examination (PSE; Wing *et al*, 1974), and if they lived in high face-to-face contact with at least one relative rated as high in expressed emotion (EE).

Of the 24 families given the opportunity to participate, only one refused, an unusual household consisting of the two brothers of a patient. Once a family had accepted, they were randomly assigned to either education plus a relatives' group or education plus family sessions. The two education sessions were given to the relatives in their home, usually while the patient was still in hospital. The relatives' group was held once a fortnight throughout the trial and was open: relatives joined when they entered the trial and were invited to attend for at least nine months. Family sessions were scheduled for once a fortnight initially and continued at this frequency for the first nine months for most families in this stream. For a few families, the sessions were spaced at monthly intervals towards the latter half of the nine months. After nine months sessions were spaced at even greater intervals for some families and ceased after the two-year follow-up for most. However, for a few families who were found to be still high in EE and in contact with the patient, sessions were continued for a maximum of three years. Family sessions lasted about one hour, and group meetings an hour and a half. An account of the management of the group is given by Berkowitz *et al* (1981), while our clinical approach to families is described by Berkowitz *et al* (1991). It shares much in common with that documented by Anderson *et al* (1986).

The initial assessments of patients and relatives were conducted by the therapists (JL, RB, NS, AS), but all follow-up assessments were made by independent raters, who were blind to group assignment. When the Camberwell Family Interview (CFI) was given at follow-up, some relatives referred to home visits by the therapists, although the rater could not tell whether these were for the education programme, which all relatives received, or for family sessions. No more than two relatives mentioned the group directly, thereby dispelling the blindness of the rater. The patients' clinical course was monitored by a psychiatrist (IG), who repeated the PSE at nine months and at two years, or at any intervening relapse. The relatives were reassessed with the CFI at the same follow-up times by a psychologist (CV). As far as we are aware, this is the first time that a two-year follow-up of EE ratings has been reported. Data from the time budget of the CFI, from other follow-up assessments, and from the therapists' regular written reports about the families have been used to monitor change in patients' social and occupational activities. This material is quantitative rather than qualitative in nature.

In this paper we present the results of the two-year follow-up of the second trial, and in addition an analysis of the combined data from the two trials.

Results

Trial of family therapy v. relatives' group

The initial assessments are reported in detail by Leff *et al* (1989). To summarise briefly, 12 families were assigned to family therapy and 11 to the relatives' group. The patients in the two streams did not differ significantly with respect to: sex, age, living group, education, marital status, drop in sociosexual achievement, occupation, duration of unemployment, presence of abnormal pre-morbid personality, number of previous admissions, age at first onset, nature of household (parental/other), relatives' ratings on EE component scales. Delusions of persecution were commoner ($P=0.024$) in patients in the family-therapy stream, but as over 50 comparisons were made this is likely to have occurred by chance. Thus randomisation effectively assigned the subjects to two groups which did not differ on a large number of characteristics, including those known to be associated with the outcome of schizophrenia.

Relatives in the family therapy stream received a median of 17 (range 8–26) sessions in the first nine months. Between the nine-month and two-year follow-up, the range was 0–16 with a median of 12. Three families received no further sessions after nine months. Three families who were still in considerable distress at two years continued with family sessions beyond that time. Of the six families who attended the relatives' group at least once in the first nine months, only one continued attending beyond that point, and carried on regularly for more than two years until the group was discontinued.

Follow-up of relatives

At the nine-month follow-up, the CFI was given to 27 of the 32 relatives. At the two-year follow-up one additional relative, who failed to attend the group, was not interviewed. However, one relative who refused the first follow-up agreed to the second, with the result that data from 27 relatives were also obtained at the two-year follow-up. All relatives assigned to family therapy were successfully followed up at this point.

The changes recorded in EE ratings and in face-to-face contact are shown in Table I, using the standard criteria for categorising each rating as high or low, and including all relatives assessed at each point in time. In any statistical comparisons, only the 26 relatives who were interviewed on all three occasions have been included.

At the nine-month follow-up (Leff *et al*, 1989) we noted significant reductions in critical comments for both streams, a significant reduction in hostility for the relatives' group, and a significant increase in warmth for both streams. There

TABLE I
Proportions of relatives showing high levels on component scales of EE and face-to-face contact initially and at the two follow-ups

	Initially	*At nine months*	*At two years*
Component scales			
Critical comments			
FT	12/16	5/15	4/16
RG	10/15	0/11	1/10
Hostility			
FT	7/16	4/15	5/16
RG	5/15	0/11	1/10
Overinvolvement			
FT	9/16	7/15	6/16
RG	10/15	6/11	2/10
Warmth			
FT	2/16	4/15	5/16
RG	1/15	4/11	3/10
Global index of EE[1]			
FT	12/12	8/11	7/12*
RG	11/11	5/9	3/8*
Contact			
FT	14/16	6/15	7/16*
RG	13/15	8/11	4/10*

FT = family therapy, RG = relatives' group.
1. The numbers refer to households.
*2 years v. initially, $P<0.05$.

was no significant reduction in overinvolvement for either stream. It is evident from Table I that between the nine-month and two-year follow-up there has been little change in the proportion of relatives in either stream scoring high on critical comments, hostility or warmth. However, a reduction in the proportion of relatives scoring high on overinvolvement has continued over time, particularly in the relatives' group. These changes are of considerable interest since shorter-term follow-ups have found over-involvement scores to be relatively stable (Brown *et al*, 1972; Leff *et al*, 1982). Of the 19 relatives rated on all three occasions, 15 were categorised as high (a score of 3 or more) initially compared with seven at two years, a significant reduction (repeated measures log-linear analysis, $F=6.8$, d.f. 2, $P=0.007$). No relative scored 5 on this scale at the two-year follow-up.

There was also a further reduction between nine months and two years in the proportion of relatives in high contact in the relatives' group stream, but not in the family therapy stream. This is of considerable importance, since analysis of the nine-month data from the two trials combined (Leff *et al*, 1989) demonstrated that family therapy was more effective in reducing contact, whereas the relatives' group exerted a greater effect on EE. The ten relatives in the relatives' group who were assessed at two years had all been in high contact initially, whereas only four remained so at the second follow-up (Fisher's exact test, $P=0.006$). All six relatives who reduced contact had attended the group at least once, while three of the four whose contact remained high were non-attenders (Fisher's exact test, $P=0.03$).

As a result of these further changes over time, by the two-year follow-up relatives in both streams showed significant reductions in EE *and* face-to-face contact compared with initial levels.

Follow-up of patients

At the nine-month follow-up it was noted that 18 of the 23 patients were compliant with their medication. One additional patient discontinued her maintenance medication between the nine-month and two-year follow-ups. Thus 74% remained on maintenance neuroleptics for two years after discharge. The comparable figures for the first trial were 83% for the experimental group and 75% for the control group.

Between the nine-month and two-year follow-ups three patients relapsed, all in the family therapy stream. Each of these relapses was type I in nature, the reappearance of psychotic symptoms in patients free of them since discharge. Two of the relapsed patients were regularly taking drugs, one oral and one depot, while one had refused medication persistently since discharge.

There were no suicides during this trial, in contrast to the first trial, but one of the relapsed patients, who was placed in a foster family, impulsively and unexpectedly jumped out of a first-floor window, breaking an ankle. The two-year relapse rates were 4 out of 12 (33%) in the family therapy stream, and 4 out of 11 (36%) in the relatives group stream. The number of patients non-compliant with medication is too small in each stream to analyse separately. This issue is addressed in the analysis of combined data from the two trials (below). With regard to compliance with the social intervention, all families assigned to family therapy received this for the duration of the trial, with one exception (who did not relapse) where the patient objected to our visits after eight sessions. As we noted earlier (Leff *et al*, 1989), five of the 11 families assigned to the relatives' group failed to attend even once. The relapse rates in this stream remained the same at two years as they had been at nine months, namely three out of five (60%) of the non-attenders compared with one out of six (17%) of the attenders.

Our previous work has been criticised for concentrating on outcome solely in terms of relapse of positive symptoms. The PSE does not extensively cover negative symptoms, but careful attention is paid to the rating of blunting of affect. In the two streams of patients combined, 18 were examined with the PSE at the two-year follow-up. Of these, ten were rated as showing some degree of blunting initially compared with four at follow-up, a nearly significant reduction (weighted least-squares estimate, $\chi^2=3.31$, $P=0.069$).

If relatives' attitudes exert an influence on the clinical course of schizophrenia, we would expect an association between changes in positive and negative symptoms and improvements in EE and contact. The actual relationships are shown in Table II. There is no suggestion that lowering face-to-face contact by itself is associated with a reduced relapse rate. The situation regarding a reduction in EE is

TABLE II
The relationships between relapse of schizophrenia over two years and reductions in EE and face-to-face contact

	Relapse[1]	*Non-relapse*[2]	*Significance*
Low contact	3	8	
High contact	4	6	NS
Low EE	1	7	
High EE	6	7	NS
Low contact plus low EE	0	6	
Others	7	8	exact $P = 0.055$

1. CFI data missing for one patient.
2. CFI data missing for one patient.

complicated by the single relapse that occurred in an apparently low-EE household. The foster mother in this household had initially been rated as high EE but had become low EE after two years of family sessions. The foster father was rated as low EE throughout. However, when the patient relapsed at the end of the two years, she accused the foster father of initiating a sexual relationship with her. This was discounted at the time because it emerged in the context of other delusional material. Since recovering from her relapse, the patient has persistently maintained that her accusation was true, and furnished details which gave it the ring of authenticity. If this were the case, the foster father should have been rated high on overinvolvement (he naturally concealed the true nature of his relationship with the patient during the repeat CFIs). This would alter the relapse rates to seven out of 14 in persistently high-EE homes and none out of seven in low-EE homes (exact $P = 0.03$). Even without this adjustment, the difference in relapse rates between households which became low in EE and low in contact and the remainder borders on significance.

Eight patients with initial blunting of affect showed no blunting at two-year follow-up. Seven of these lived in households in which EE and/or contact became low, compared with two out of four patients whose blunting remained unchanged or worsened, a non-significant difference. Data relating to social and occupational activities were obtained from the time budget of the CFI, and from the therapists' regular reports. Information was collected under the headings of living group, sexual relationships, friends, and occupation. No data were obtained for two patients in the relatives' group stream, leaving 21 patients who were included in this analysis. Between the nine-month and two-year follow-ups the following changes took place: two patients moved from their parental home to a flat, but one of them continued to spend most of his time with his parents. Two patients previously without a partner established a stable sexual relationship. Two patients increased their social activity considerably. Four patients improved their occupational status, two by finding a full-time job, one by going to college, and the other by attending day classes. With the sole exception of the patient who moved out but continued to visit his parents daily, all these improvements were shown by patients in the relatives' group stream. No deterioration in any sphere of activity was noted for the patients followed up.

Analysis of combined data from the first and second intervention trials

When reporting the nine-month data (Leff *et al*, 1989), we noted a small number of significant differences in demographic and clinical variables between the subjects in Trial 1 and Trial 2. However, as none of these was related

TABLE III
Relapse rates (excluding two suicides) in the two trials of social intervention

Trial	*Group*	n	*No. of psychotic relapses*	*(%)*	*No. of relapses including suicides*	*(%)*
1	Control	12	9	(75)[1]		
1	Package of interventions	12	4	(33)	6	(50)
2	Family therapy	12	4	(33)		
2	Relatives' group	11	4	(36)		
1+2	Any social intervention	35	12	(34)[2]	14	(40)[3]

1.v 2: $\chi^2 = 5.99$, $P < 0.02$.
1.v.3: $\chi^2 = 4\ 21$, $P < 0.05$

to outcome, we considered it justifiable to compare relapse rates across trials. The two-year outcome of the various groups is shown in Table III.

The relapse rate of patients who discontinued maintenance neuroleptics was 7 out of 11 (64%) compared with 14 out of 36 (39%) for those who stayed on drugs. This difference of 25% points to the benefit of maintenance drugs, but with these relatively small numbers of patients is non-significant ($\chi^2 = 2.23$). Therefore drug compliance has not been taken into account in comparing the relapse rates of the various groups. If the two suicides in the first trial are excluded, the relapse rates of the three groups assigned to some form of social intervention are virtually identical. To apply the most conservative test to the social interventions, we should include the suicides with the relapses and ignore the fact that some families were non-compliant, particularly with the relatives' group in Trial 2. Taking this approach, the relapse rate in families assigned to a social intervention was slightly above half that in the control group, a significant difference ($P < 0.05$).

These data are presented in the form of survival curves in Fig. 1. The difference in relapse rates over time between the two groups in Trial 2 was tested with the log rank test and the Wilcoxon rank test and found to be non-significant.

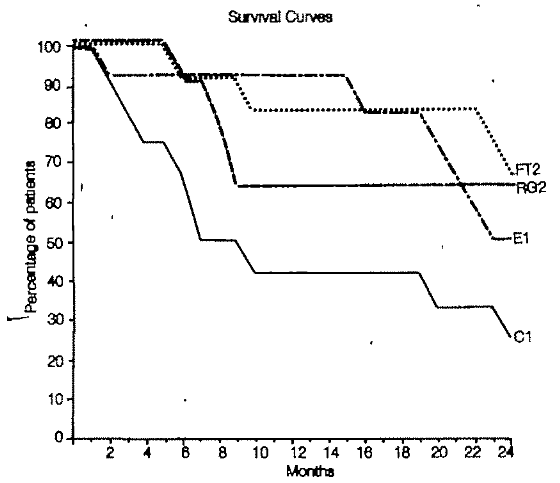


FIG. 1 Percentage of patients free of relapse over time in the two intervention trials (C1 control group, trial 1; E1 experimental group, trial 1; FT2 family therapy stream, trial 2; RG2 relatives' group stream, trial 2).

Discussion

This trial was initiated with the prime purpose of answering a resource question: which is the most efficient and effective way of delivering help to families of schizophrenic patients? The two-year findings show that the overall relapse rate of patients is very similar, and significantly lower than that of a control group, whether help is given through the medium of family sessions in the home, a relatives' group, or a combination of the two, as in the first trial. This result would point to the relatives' group as being the mode of choice, since it enables therapists to deal with a much larger case load of families. However, the high non-compliance rate in the relatives' group stream must modify this conclusion. It is evident from the follow-up data that offering the relatives' group on its own divides the families into those with a strong motivation, in which the patients do very well (17% relapse rate over two years), and those who are unable or unwilling to attend the group, in which the patients do badly (60% relapse rate over two years). Since randomisation was effective in equating the two streams for factors known to be associated with outcome, it is reasonable to assume that the family therapy stream contained a similar proportion of families who would have failed to attend the group. However, with the exception of one patient, they complied with family sessions in the home.

The majority of relapses in the drugs-only group (first trial control condition) and all the relapses in the relatives' group stream occur in the first nine months. The relapse rates in the intervention package group and the family therapy stream remain low for the first 20 months. There is then a sharp down-turn in the intervention package group, contributed to by the two suicides in addition to some relapses. It appears that if suicides can be avoided, and there were none in the second trial, the intervention package is as effective as family therapy. It is more efficient, since we learned from the first trial that a relatively small number of family sessions in the home induces most relatives to attend the group regularly. However, there will always be some relatives who will not come to the group under any circumstances, and it is essential for the therapists to be available for home visits.

In terms of EE and face-to-face contact, the two-year follow-up data shed new light on the time course of changes in these features. Major changes in critical comments, hostility and warmth were recorded at nine months, but they then remained fairly stable up to two years. By contrast overinvolvement showed a non-significant decline by nine months, which continued up to two years, reaching significance at that point. We cannot ascribe this reduction to the interventions with any certainty, since two-year follow-up data are not available for any naturalistic sample of relatives. However, it is of great interest that overinvolvement declines much more slowly than the other negative EE components, supporting our view that it is an attitude formed over a much longer period.

The reduction in face-to-face contact noted in the relatives' group stream, particularly among the attenders, has important implications for a systemic view of family functioning. It demonstrates that significant changes in the emotional attitudes of relatives and in their interaction with the patient can occur without the patient being directly involved in the therapy. It also shows that over time both forms of intervention were associated with significant changes in *both* social factors targeted in our interventions.

The main impetus behind our trials was to explore whether the associations found in the naturalistic studies could be explained by a direct influence of interpersonal relations on the course of schizophrenia. We have presented evidence elsewhere (Leff, 1989) that therapeutically induced reductions in relatives' EE and/or contact lead to a lowered relapse rate over nine months. Our two-year follow-up data do not entirely support this proposition. There is a problem in interpreting the data owing to a probable misclassification of the EE level of one household. If this is taken into account, reduction in EE is associated with a lowered relapse rate over two years. An alternative interpretation is that for patients to remain free of relapse over two years it is necessary for *both* EE and contact to be reduced. In the six households showing this combination, no patient relapsed. However, this inference must remain tentative at present, since the result was on the borderline of significance, and no two-year follow-up data exist from the naturalistic studies which could be used to test it.

Turning to the outcome measures other than relapse, it was gratifying to discover a near-significant reduction in blunting of affect over two years. In the absence of a control group we cannot be certain that this was a consequence of the interventions. However, it is important to have detected a decline in this negative symptom, since relatives' critical comments are directed predominantly at negative rather than at positive symptoms (Leff & Vaughn, 1985). There were no dramatic improvements in social functioning, although as we were only recording quantitative data, we could have missed important qualitative changes. On the other hand, the changes we detected were all positive and would confer long-term benefits on the small number of patients involved. In particular, four patients moved out of the parental home to live independently, and to our knowledge none has returned to date.

In the introduction to this paper we stressed that four other research groups have conducted trials with similar designs and outcomes to ours. All have reported on a two-year follow-up, except the pioneering study of Goldstein *et al* (1978) in which an uncontrolled follow-up of between three and five years was carried out. In the others, the two-year relapse rates for patients in families receiving a form of intervention were 17% (Falloon *et al*, 1985), 25% (Hogarty *et al*, 1987) and 33% (Tarrier *et al*, 1989). Our own rates of 33% for family therapy and 36% for the relatives' group are among the highest of these results, but then we selected patients at highest risk of relapse by virtue of being in high contact with high-EE relatives. None of the other studies included high contact as a selection criterion.

It is evident that the results of these studies cannot be extrapolated to schizophrenic patients living in low-EE households or on their own. However, they indicate that for patients living with high-EE relatives, who are at high risk of relapse, family intervention combined with maintenance neuroleptics confers a significant advantage in terms of outcome over two years. Our own data lead us to endorse the recommendation in our previous paper (Leff *et al*, 1989) that relatives' groups be set up in conjunction with family sessions in the home. For the majority of relatives, a few home sessions are sufficient to maintain regular attendance at the group. The data presented here show that even if relatives attend for no more than nine months, significant reductions in EE and face-to-face contact can be expected. There remains an outstanding issue of whether the interventions we are recommending can be effectively delivered by staff less highly trained and motivated than those participating in these research studies. This is an empirical question which we intend to address in our next study.

Acknowledgements

We are grateful to Mr G Der for statistical advice and for performing many of the analyses reported in this paper.

References

ANDERSON, C. M., REISS, D. J. & HOGARTY, G. E. (1986) *Schizophrenia in the Family: A Practitioner's Guide to Psychoeducation and Management*. New York: Guilford Press.

BERKOWITZ, R., KUIPERS, L., EBERLEIN-FRIES, R., *et al* (1981) Lowering expressed emotion in relatives of schizophrenics. In *New Developments in Interventions with Families of Schizophrenics* (ed. M. J. Goldstein). London: Jossey-Bass.

——, SHAVIT, N. & LEFF, J. (1991) Psychosocial interventions with schizophrenic patients and their families. *Family Process* (in press)

BROWN, G. W., BIRLEY, J. L. T. & WING, J. K. (1972) Influence of family life on the course of schizophrenic disorders: a replication *British Journal of Psychiatry*, **121**, 241–258.

DULZ, B. & HAND, I. (1986) Short-term relapse in young schizophrenics. can it be predicted and affected by family (CFI), patient and treatment variables? In *Treatment of Schizophrenia* (eds M. J. Goldstein, I. Hand & K Hahlweg). Berlin: Springer-Verlag.

FALLOON, I. R. H, BOYD, J. L., MCGILL, C. W., *et al* (1982) Family management in the prevention of exacerbations of schizophrenia. *New England Journal of Medicine*, **306**, 1437-1440.

——, WILLIAMSON, M., RAZANI, J., *et al* (1985) Family versus individual management in the prevention of morbidity of schizophrenia: I. Clinical outcome of a two-year controlled study. *Archives of General Psychiatry*, **42**, 887-896.

GOLDSTEIN, M. J., RODNICK, E. H., EVANS, J. R., *et al* (1978) Drug and family therapy in the aftercare treatment of acute schizophrenia. *Archives of General Psychiatry*, **35**, 169-177.

HOGARTY, G. E., ANDERSON, C. M., REISS, D. J., *et al* (1986) Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia: I. One-year effects of a controlled study on relapse and expressed emotion. *Archives of General Psychiatry*, **43**, 633-642.

——, —— & —— (1987) Family psychoeducation, social skills training and medication in schizophrenia: the long and the short of it. *Psychopharmacology Bulletin*, **23**, 12-13.

LEFF, J. P. (1985) Family treatment of schizophrenia. In *Recent Advances in Clinical Psychiatry* (ed. K. Granville-Grossman), no. 5. London: Churchill Livingstone.

—— (1989) Controversial issues and growing points in research on relatives' expressed emotion. *International Journal of Social Psychiatry*, **35**, 133-145.

——, KUIPERS, L., BERKOWITZ, R., *et al* (1982) A controlled trial of intervention in the families of schizophrenic patients. *British Journal of Psychiatry*, **141**, 121-134.

——, ——, —— *et al* (1985) A controlled trial of social intervention in the families of schizophrenic patients: two-year follow-up. *British Journal of Psychiatry*, **146**, 594-600.

—— & VAUGHN, C. (1985) *Expressed Emotion in Families*. New York: Guilford Press.

——, BERKOWITZ, R., SHAVIT, N., *et al* (1989) A trial of family therapy v. a relatives group for schizophrenia. *British Journal of Psychiatry*, **154**, 58-66.

STRACHAN, A. M. (1986) Family intervention for the rehabilitation of schizophrenia: toward protection and coping. *Schizophrenia Bulletin*, **12**, 678-698.

TARRIER, N., BARROWCLOUGH, C., VAUGHN, C., *et al* (1988) The community management of schizophrenia: a controlled trial of a behavioural intervention with families to reduce relapse. *British Journal of Psychiatry*, **153**, 532-542.

——, ——, ——, *et al* (1989) Community management of schizophrenia: a two-year follow-up of a behavioural intervention with families. *British Journal of Psychiatry*, **154**, 625-628.

WING, J. K., COOPER, J. E. & SARTORIUS, N. (1974) *Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

*Julian Leff, BSc, MD, FRCPsych, MRCP, *Professor of Social and Cultural Psychiatry, Director, MRC Social and Community Psychiatry Unit, Institute of Psychiatry, De Crespigny Park, London SE5 8AF*; Ruth Berkowitz, BA, BSc, PhD, *Research Associate*; Naomi Shavit, MA, *Research Associate*; Angus Strachan, PhD, *Adjunct Assistant Professor of Psychology, University of California, Los Angeles*; Ilana Glass, MA, MB, MRCPsych, MPhil, *Research Psychiatrist*; Christine Vaughn, PhD, *Scientific Staff*

*Correspondence

British Journal of Psychiatry (1990), 157, 578–584

Obstetric Complications, the Putative Familial–Sporadic Distinction, and Tardive Dyskinesia in Schizophrenia

EADBHARD O'CALLAGHAN, CONALL LARKIN, ANTHONY KINSELLA and JOHN L. WADDINGTON

Obstetric complications were more common in the histories of those schizophrenic out-patients without a family history of psychiatric disorder, and were associated with an earlier onset of their illness. Those patients with tardive dyskinesia were more likely to have a family history of psychiatric disorder, less likely to have experienced obstetric complications, and showed greater cognitive deficit. Obstetric complications should be considered in juxtaposition with genetic factors in evaluating the putative familial–sporadic distinction in schizophrenia. Additionally, familial/genetic factors appear to contribute to vulnerability to tardive dyskinesia.

There has recently been a resurgence of interest in the familial–sporadic distinction in schizophrenia as a research strategy (Eaves *et al*, 1986; Lewis *et al*, 1987). This proposes that schizophrenic patients might usefully be subdivided into those with (i.e. familial) and those without (i.e. sporadic) one or more affected relatives; these subgroups may then differ in clinical characteristics and potential risk factors, and thus provide important clues to aetiological heterogeneity. In juxtaposition is the debate as to whether sporadic cases might be characterised by an excess of environmental insults resulting in cerebral dysfunction. One form of such insult is obstetric complications (OCs), which occur to excess in the histories of schizophrenic patients (Lewis & Murray, 1987; McNeill & Kaij, 1978). Their clinical correlates remain ill-defined, and they have been incorporated into a diverse range of aetiological schemes: for example, it has been proposed that OCs may add to the weight of liability within a multifactorial/threshold model (Parnas *et al*, 1982), or that they might initiate periventricular haemorrhaging and resultant structural dysplasia or abnormal neuronal circuitry as a specific pathophysiological process leading to schizophrenia (Lewis & Murray, 1987).

Integration of OCs within the putative familial–sporadic distinction leads to the hypothesis that such events may be over-represented in sporadic cases and under-represented in familial cases. However, this theory and preliminary results supporting it (Lewis & Murray, 1987) have attracted both criticism (Goldin *et al*, 1987; McGuffin *et al*, 1987) and contradictory data (Nimgaonkar *et al*, 1988). This may stem from a number of methodological problems: firstly, unless the family is both large and systematically evaluated, it may not be possible to classify each patient accurately as a familial or a sporadic case; secondly, it is not clear what constitutes the most appropriate definition of a positive family history, either in terms of the spectrum of disorders to be included or their degree of relatedness to the patient; and thirdly, the retrospective determination of OCs, whether from patient records or from maternal interview, is of unknown validity.

Clarification of these issues may be important in relation to another aspect of schizophrenia. There is now some weight of evidence to indicate that schizophrenic patients with tardive dyskinesia, especially that with an orofacial topography, are more likely to show deficits in a variety of cognitive functions than those without such movement disorder (Waddington, 1987; Waddington *et al*, 1987). One interpretation of this is that patients with organic brain dysfunction might be more likely to develop tardive dyskinesia during long-term neuroleptic treatment; OCs might be one potential source of such cerebral dysfunction and thus predispose to the emergence of tardive dyskinesia (Waddington & Youssef, 1987).

The present study was undertaken to investigate potential clinical correlates of OCs in relation to the putative familial–sporadic distinction in schizophrenia, and to explore whether they were associated with raised vulnerability to tardive dyskinesia.

Method

The subjects were out-patients attending the depot neuroleptic clinic of Cluain Mhuire Family Centre, Blackrock, Co. Dublin. Of 75 unselected attendees evaluated using the Abnormal Involuntary Movement Scale (AIMS; National Institute of Mental Health, 1976), 64 were subsequently found to satisfy DSM–III criteria (American Psychiatric Association, 1980) for schizophrenia on the basis of case-note review and, where necessary, clinical interview.

TABLE I

Characteristics of obstetric complications recorded from maternal interview in 15 of 45 cases

Case	*Obstetric history*
1.	Antepartum haemorrhage; severe pre-eclampsia; incubated for >4 weeks
2.	Antepartum haemorrhage; severe pre-eclampsia
3.	Multiple antepartum haemorrhages and transfusions; premature (<37 weeks); incubated for >4 weeks
4.	Born into lavatory bowl; cord not cut for >1 hour; 'blue' at birth; in hospital for 2 months
5.	Cord prolapse; abnormal presentation; incubated for 6 weeks
6.	Severe pre-eclampsia; post-mature (>42 weeks); breech; in labour for >48 hours
7.	Post-mature (>42 weeks); cord prolapse; high forceps; incubated for >4 weeks
8.	Post-mature (>42 weeks); high forceps
9.	Antepartum haemorrhage; premature (<37 weeks); breech; incubated for >4 weeks
10.	Post-mature (>42 weeks); long labour (>48 hours); high forceps
11.	Breech; high forceps
12.	Cord prolapse; incubated for 6 weeks
13.	Cord prolapse
14.	Cord prolapse
15.	Birthweight <2000 g; incubated for 4 weeks

Sixty-three of the patients consented both to their own participation in the study and to an approach being made to their mother; the remaining one refused such consent. For 17 of these patients, their biological mother was deceased or the patient had been adopted. Thus, the final study population consisted of 45 patients. There were 29 men and 16 women, whose mean (s.d.) age was 32.3 (7.3) years (range 20–48) and who had been ill for 9.5 (5.2) years (range 1–23).

For each patient, demographic information and clinical history were obtained from their case records. Cognitive function was evaluated at the time of AIMS assessment using Trail Making Tests A and B from the Halstead–Reitan battery, two visual scanning, visuomotor co-ordination and cognitive flexibility tasks sensitive to general organic brain dysfunction (Reitan, 1958; Wegner *et al*, 1985). The time in seconds required to complete each test was recorded for all patients except one who did not complete neuropsychological evaluation.

Initial contact with each patient's biological mother was made by mail and followed up where possible by telephone. After the purpose of the study had been explained, each consented to be interviewed. In all instances but one this was a personal interview; in the remaining instance the woman lived in a remote region of Ireland, but took part in a series of telephone interviews from a public call-box.

These interviews had two distinct elements, relating to obstetric and to family psychiatric history. Firstly, each mother was asked about the place of the patient's birth and his/her position in the birth order (1 = first born). Details were recorded of any injury, illness or exposure to drugs during the pregnancy. She was then taken through her obstetric history in relation to the patient using the OC Scale of Lewis & Murray (Lewis *et al*, 1989; see also Nimgaonkar *et al*, 1988). At least one *definite* complication by this scale was required for allocation to the group who had experienced OCs; absence of such occurrences, or the presence only of 'equivocal' signs, resulted in allocation to the group who had not experienced OCs.

The second element of each interview followed Baker *et al*'s (1987) procedure. This involved the construction of a family tree of at least all first- and second-degree relatives of the patient, and a life history being taken for each relative for specification of any psychiatric disorder according to the family history method. Two criteria of a positive family history were adopted (Lewis *et al*, 1987; Nimgaonkar *et al*, 1988): narrow, defined by a first-degree relative with schizophrenia according to Family/History–Research Diagnostic Criteria (Andreasen *et al*, 1977); and broad, defined by a first- or second-degree relative with schizophrenia or affective disorder, or who had committed suicide.

Results

From maternal interviews, *definite* OCs had been present in relation to 15 (33%) of the 45 patients, and these are detailed in Table I. The most commonly reported OCs were antepartum haemorrhage (four cases), cord prolapse (five cases) and post-maturity (four cases); the seven instances of a prolonged period in an incubator following delivery are most likely secondary, reflecting the consequences of the earlier events indicated. In relation to only one patient (case 4, Table I) were the circumstances of the delivery so unusual as to defy the criteria of the OC Scale used, but by any standard of obstetric practice they constituted complications.

We were able to locate the maternity hospital records relating to the pregnancies which resulted in 21 of the 75 patients originally screened for an item-by-item comparison with maternal recall (O'Callaghan *et al*, 1990). Regarding the subgroup of 17 of the present 45 mothers for whom maternity hospital records were available, there was complete concordance (Cohen, 1960) for the absence of OCs ($n = 10$) or the presence ($n = 7$) of at least one common *definite* OC on application of the Scale to each source of information (kappa = 1.0, $Z = 4.12$; $P < 0.001$).

TABLE II
Number of patients with a history of obstetric complications in relation to family history of psychiatric disorder

Family history of psychiatric disorder	Obstetric complications Absent	Present
Narrow criterion[1]		
Absent	16	13
Present	14	2
Broad criterion[2]		
Absent	6	9
Present	24	6

1. $\chi^2=3.50$, $P<0.065$.
2. $\chi^2=5.51$, $P<0.02$.

As shown in Table II, there was a significant dissociation between the presence of OCs and of a family history of psychiatric disorder: OCs were more common in the histories of those patients without a family history. There was a tendency towards such a dissociation using the narrow criterion of family history, but this became more robust statistically when the broad criterion was applied.

The 15 patients who had experienced OCs and the 30 patients who had not experienced such events were of indistinguishable age and sex distribution (Table III). However, the former showed a significantly younger age at onset of their illness, in terms of their first contact with a psychiatric service. These two groups did not appear to differ on univariate comparison of position in birth order, number of siblings, or performance on Trail Making Tests A and B.

These data were entered into stepwise multiple logistic regression analysis (Dixon, 1983; Fleiss *et al*, 1986) to construct a hierarchy of those variables having independent associations with the binary measure of the presence or absence of a history of OCs; this stepwise algorithm generates a 'best' final model which takes into account any intercorrelated measures, and its adequacy was confirmed using the goodness-of-fit tests of Hosmer & Lemeshow and Brown (see Waddington *et al*, 1987). As shown in Table IV, the final logistic model confirmed the presence of OCs to be associated with earlier age at onset of illness, and additionally suggested some association with a lower position in the birth order.

Tardive dyskinesia, OCs and family history

There were 12 patients (27%) who showed at least one AIMS score of two (mild) on one orofacial region; in fact, each of these 12 also satisfied the more stringent criteria of Schooler & Kane (1982) when applied solely to the orofacial area (i.e. at least two AIMS scores of two on two regions or at least one score of three (moderate) on one region).

As shown in Table V, there was a significant dissociation between the presence of tardive dyskinesia and a history of OCs: those patients with such movement disorder were less likely to have experienced OCs. Also, there was a significant association between the presence of tardive dyskinesia and of a family history of psychiatric disorder. There was a tendency towards such an association using the broad criterion of family history, but this became more robust statistically when the narrow criterion was applied. Eight of the 12 cases of tardive dyskinesia (66%) were among the 14 patients who had such a family history of psychiatric disorder *without* a history of OCs; the remaining four cases (33%) were among the 16 patients who had neither a family psychiatric history nor a history of OCs; there were no cases among the 15 patients with a history of OCs, either with ($n=2$) or without ($n=13$) a family history of psychiatric disorder ($\chi^2=12.14$, d.f. $=2$, $P<0.005$).

The 12 patients with and the 33 without tardive dyskinesia were of indistinguishable age and sex distribution, age at onset of illness, and birth order (Table III). However, those with this movement disorder showed significantly poorer cognitive function; this was most marked in terms of performance on Trail Making Test B, which places an additional demand on cognitive flexibility. Curiously, patients with tardive dyskinesia had a larger number of siblings, and this effect was confirmed on application of the Greenwood-Yule procedure (Ernst & Angst, 1983).

These data were entered into stepwise multiple logistic-regression analysis to construct a hierarchy of those

TABLE III
Clinical correlates of obstetric complications and tardive dyskinesia

Variable	Obstetric complications Absent	Obstetric complications Present	Tardive dyskinesia Absent	Tardive dyskinesia Present
Number of subjects (men/women)	30(21/9)	15 (8/7)	33(21/12)	12 (8/4)
Mean (s.d.) age: years	33.4(8.0)	30.3(5.5)	31.1(7.7)	35.7(5.2)
Mean (s.d.) age at onset[1]: years	24.0(5.5)	20.6(4.8)*	22.3(5.9)	24.4(3.9)
Mean (s.d.) position in birth order	3.4(2.4)	2.5(1.7)	2.8(2.1)	3.9(2.5)
Mean (s.d.) no. of siblings	4.8(2.7)	4.2(2.3)	4.1(2.2)	6.0(3.1)*
Mean (s.d.) time for trail making test A:s	53.6(25.2)	44.4(12.8)	45.8(13.8)	63.6(33.9)*
Mean (s.d.) time for trail making test B:s	129.2(61.3)	103.7(53.1)	101.4(41.3)	173.5(69.9)**

$^*P<0.05$, $^{**}P<0.001$, Student's *t*-tests.
1. Defined in terms of first contact with psychiatric service.

TABLE IV

Hierarchies of variables related by logistic regression analysis to the presence or absence of (a) obstetric complications and (b) tardive dyskinesia

Variable	β	$SE\beta$	$\beta/SE\beta$	χ^2	P
Obstetric complications[1]					
Age at onset	−0.163	0.074	−2.21	3.74	0.053
Birth order	−0.330	0.206	−1.60	3.27	0.071
Tardive dyskinesia[1]					
Trail making test B	0.031	0.012	2.60	13.30	<0.001
No. of siblings	0.511	0.249	2.05	4.43	<0.05

1. Goodness-of-fit tests: (a) Hosmer, $\chi^2=5.54$, $P=0.70$, Brown, $\chi^2=2.71$, $P=0.26$, (b) Hosmer, $\chi^2=3.79$, $P=0.88$; Brown, $\chi^2=0.46$, $P=0.79$.

variables having independent associations with the binary measure of the presence or absence of tardive dyskinesia. As shown in Table IV, the goodness of fit of the final logistic model was satisfactory, and it confirmed that poor performance on Trail Making Test B was the primary variable associated with tardive dyskinesia. It also confirmed that number of siblings was modestly associated with the presence of such movement disorder.

TABLE V

Number of patients with tardive dyskinesia in relation to obstetric complications and family history of psychiatric disorder (narrow and broad criteria)

	Tardive dyskinesia	
	Absent	*Present*
Obstetric complications[1]		
Absent	18	12
Present	15	0
Narrow criterion of family psychiatric history[2]		
Absent	25	4
Present	8	8
Broad criterion of family psychiatric history[3]		
Absent	14	1
Present	19	11

1. $\chi^2=6.26$, $P<0.02$.
2. $\chi^2=5.18$, $P<0.025$.
3. $\chi^2=3.20$, $P<0.075$.

Discussion

In this study we applied Family/History–Research Diagnostic Criteria, shown to improve the sensitivity, reliability and validity of the family-history method (Andreasen *et al*, 1977, 1986), to information derived from interview with each patient's biological mother. An additional procedure, the construction of a family tree with individual life histories, has been shown to further increase the sensitivity of this approach (Baker *et al*, 1987). The generally large family size in Ireland (e.g. here, a mean of five siblings per patient) gave our study further power to specify the presence or absence of a family history of psychiatric disorder. We adopted the strategy that both narrow and broad definitions of a family history of psychiatric disorder should be used in tandem (Lewis *et al*, 1987). Regarding our OC data, we used a conventional maternal interview approach. The extent of concordance for OC status between maternal recall and maternity hospital records for a subgroup of these mothers suggests that our overall categorisation of patients on the basis of maternal recall has validity (O'Callaghan *et al*, 1990).

It has been shown that schizophrenic patients are more likely to have experienced OCs than are other psychiatric patients or normal subjects (McNeil, 1987; Lewis & Murray, 1987). We found that a history of OCs was significantly more likely to occur in schizophrenic patients *without* a family history of psychiatric disorder, and vice versa. Such a relationship can be predicted on theoretical grounds (McNeil & Kaij, 1978), but recent studies which have addressed the issue have proved inconclusive (Lewis & Murray, 1987; Nimgaonkar *et al*, 1988). Perhaps the use of methodological refinements increased our power to detect it.

This inverse relationship of OC and family history of psychiatric disorder would appear consistent with both classic 'stress-diathesis' and 'organic phenocopy' hypotheses for the aetiology of schizophrenia. Regarding the former, our data concern the extent of putative genetic loading between groups rather than within individuals, but might be accounted for in such terms. Regarding the latter, it has been proposed that schizophrenic patients without a family psychiatric history but with a history of OCs are an aetiologically distinct subgroup that is more likely to show developmentally determined abnormalities of cerebral morphology on computerised tomography (CT) (Lewis & Murray, 1987), but there are contradictory data (Owen *et al*, 1988; Nimgaonkar *et al*, 1988). Our failure to find any differences in cognitive function between patients with and without a history of OCs using a neuropsychological test of established sensitivity to organic brain dysfunction (and able to readily distinguish patients with and without tardive dyskinesia) could be interpreted as inconsistent with such a proposition. However, the relationship between cognitive dysfunction and structural brain pathology in schizophrenia appears much more complex than originally envisaged (Zec & Weinberger, 1986), and our neuropsychological tests must be regarded as far from definitive.

Regarding non-familial correlates of OCs, patients with a history of such events showed a younger age at onset of their schizophrenic illness, and this was evident using both conventional and logistic regression analyses; similar findings have been noted by Owen *et al* (1988). Variability in age at onset of schizophrenia is well recognised but little understood. A review of the relevant literature (Kendler *et al*, 1987) revealed no strong or reliable relationship of age at onset to risk either for schizophrenia or for other psychiatric disorders in relatives. Therefore, environmental factors such as OCs, rather than (or in addition to) putative genetic liability, may influence this illness characteristic. OCs may themselves be the result of other aetiologically relevant processes, and are a heterogeneous group of events that might be expected to have a variety of effects on the foetus; however, the common pathophysiological process that is most widely entertained, which possibly results in subtle cerebral dysfunction, is cerebral anoxia (McNeil, 1987). Such early cerebral damage might synergise with other aetiological factors, not just to increase the overall likelihood of schizophrenia emerging but perhaps also to precipitate its onset (O'Callaghan *et al*, 1988). Using logistic regression analysis, we additionally found an early position in the birth order to have some statistical association with a history of OCs. This would be consistent with the general body of evidence (Ernst & Angst, 1983) that those born prematurely may be at particular risk for unfavourable biological events during pregnancy and birth.

Our other main finding was that tardive dyskinesia was more likely to emerge in patients with a stronger familial component to their illness. We also replicated previous reports (see Waddington, 1987) that patients with tardive dyskinesia, especially that with an orofacial topography (Waddington *et al*, 1987), are more likely to show cognitive dysfunction. On the basis of the putative relationship both of the occurrence of OCs in the absence of a family psychiatric history (Lewis & Murray, 1987) and of poor performance on neuropsychological tests (Zec & Weinberger, 1986) to structural brain pathology, we had predicted that patients with a history of OCs might be particularly vulnerable to tardive dyskinesia (Waddington & Youssef, 1987). However, our data were not only inconsistent with this notion, but pointed clearly in the opposite direction; indeed, patients *with* a family history in the *absence* of OCs appeared most at risk for such movement disorder.

Regarding familial aspects of tardive dyskinesia, concordance for its presence has been reported in one schizophrenic sibling pair, and concordance for its absence noted in each of four other sibling pairs, including one set of twins (Yassa & Ananth, 1981). While Bartels *et al* (1985) reported a reduced likelihood of a family history of schizophrenia in patients with tardive dyskinesia, the in-patient population of that study was some 30 years older than our own out-patients, and assessment of family history was dissimilar. Conversely, Wegner *et al* (1985) reported no difference in the likelihood of a family history of schizophrenia between younger schizophrenic and schizoaffective out-patients with and without tardive dyskinesia. Concordance for tardive dyskinesia has been noted in two schizophrenic brothers, one showing mild and the other marked cognitive dysfunction (Weinhold *et al*, 1981), and we have reported elsewhere concordance for tardive dyskinesia in a rare group of four schizophrenic siblings, each with cognitive impairment (Waddington & Youssef, 1988). Thus, there is some support for an association between tardive dyskinesia and putative genetic loading for schizophrenia.

The greater cognitive deficit (and by implication the more neurological abnormality) that appears to be evident in patients with tardive dyskinesia might be at least in part related to the more familial nature of their illness. Cognitive function has indeed been reported to be more impaired in schizophrenic patients with a family history of psychiatric disorder (Orzack & Kornetsky, 1971; Asarnow *et al*, 1978; Walker & Shaye, 1982), and, while we have no ready explanation for our finding of an association between tardive dyskinesia and having a larger number of siblings, Orzack & Kornetsky (1971) noted that increasing numbers of siblings is predictive of poor cognitive function. Additionally, patients with a family history of schizophrenia show more neurological soft signs (Walker & Shaye, 1982). There is evidence that manifestation of such signs, reported to occur to excess in patients with tardive dyskinesia (Wegner *et al*, 1985), is a familially transmitted abnormality of neurological state in schizophrenia (Kinney *et al*, 1986).

In longitudinal studies, pre-schizophrenic infants have shown dysregulation of neurological and cognitive development (Fish, 1977, 1987); this was related to the severity of later psychiatric disorder, and was associated with the triad of correlates of tardive dyskinesia encountered in our study (i.e. a positive family history of schizophrenia, independence from OCs, and cognitive disorder). It may be that a component of vulnerability to tardive dyskinesia resides in cerebral dysfunction that is determined very early in neurodevelopment. Our finding of an excess release of developmental reflexes in schizophrenic and bipolar patients with tardive dyskinesia (Youssef & Waddington, 1988*a*) and of greater developmental deficit in mentally handicapped patients with tardive dyskinesia (Youssef & Waddington, 1988*b*) would be consistent with this notion.

In summary, we report that schizophrenic patients with a history of OCs appear less likely to have a positive family history of psychiatric disorder, and have a younger age at onset of their illness; such data extend the scope of current neurodevelopmental perspectives of schizophrenia (Weinberger, 1987; Murray & Lewis, 1987; Waddington, 1988) by indicating that OCs should be considered in juxtaposition with genetic influences. Additionally, familial/genetic factors appear to contribute not only to the aetiology of schizophrenia but also to vulnerability to tardive dyskinesia.

Acknowledgements

This work was supported by the Order of St John of God. It was greatly aided by the kind co-operation of mothers of patients attending Cluain Mhuire Family Centre, through the local branch of the Schizophrenia Association of Ireland. We thank Drs S. W. Lewis and R. M. Murray for making their Obstetric Complications Scale available to us.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn). Washington, DC: APA.

ANDREASEN, N. C., ENDICOTT, J., SPITZER, R., *et al* (1977) The family history method using diagnostic criteria. *Archives of General Psychiatry*, **34**, 1229–1235.

——, RICE, J., ENDICOTT, J., *et al* (1986) The family history approach to diagnosis: how useful is it? *Archives of General Psychiatry*, **43**, 421–429.

ASARNOW, R. T., CROMWELL, R. L. & RENNICK, P. M. (1978) Cognitive and evoked response measures of information processing in schizophrenics with and without a family history of schizophrenia. *Journal of Nervous and Mental Disease*, **166**, 719–730.

BAKER, N. J., BERRY, S. L. & ADLER, L. E. (1987) Family diagnoses missed on a clinical inpatient service. *American Journal of Psychiatry*, **144**, 630–632

BARTELS, M., MANN, K. & FRIEDRICH, W. (1985) Tardive dyskinesia: marked predominance of non-genetic schizophrenia. *Biological Psychiatry*, **20**, 101–103.

COHEN, J. (1960) A coefficient of agreement for nominal scales *Educational and Psychological Measurement*, **20**, 37–46.

DIXON, W. J. (1983) *BMDP Statistical Software*. Berkeley: University of California Press.

EAVES, L. J., KENDLER, K. S. & SCHULZ, S. C. (1986) The familial-sporadic classification: its power for the resolution of genetic and environmental etiologic factors. *Journal of Psychiatric Research*, **20**, 115–130.

ERNST, C. & ANGST, J. (1983) *Birth Order*. Berlin: Springer Verlag.

FISH, B. (1977) Neurobiologic antecedents of schizophrenia in children: evidence for an inherited, congenital neurointegrative defect. *Archives of General Psychiatry*, **34**, 1297–1313.

—— (1987) Infant predictors of the longitudinal course of schizophrenic development. *Schizophrenia Bulletin*, **13**, 395–409.

FLEISS, J. L., WILLIAMS, J. B. W. & DUBRO, A. F. (1986) Logistic regression analysis of psychiatric data. *Journal of Psychiatric Research*, **20**, 195–209.

GOLDIN, L. R., DELISI, L. E. & GERSHON, E. S. (1987) Unravelling the relationship between genetic and environmental risk factors in psychiatric disorders. *British Journal of Psychiatry*, **151**, 302–305.

KENDLER, K. S., TSUANG, M. T. & HAYS, P. (1987) Age at onset in schizophrenia: a familial perspective. *Archives of General Psychiatry*, **44**, 881–890.

KINNEY, D. K., WOODS, B. T. & YURGELUN-TODD, D. (1986) Neurologic abnormalities in schizophrenic patients and their families. II. Neurological and psychiatric findings in relatives. *Archives of General Psychiatry*, **43**, 665–668.

LEWIS, S. W. & MURRAY, R. M. (1987) Obstetric complications, neurodevelopmental deviance and risk of schizophrenia. *Journal of Psychiatric Research*, **21**, 413–422.

——, REVELEY, A. M., REVELEY, M. A., *et al* (1987) The familial/sporadic distinction as a strategy in schizophrenia research. *British Journal of Psychiatry*, **151**, 306–313.

——, OWEN, M. J. & MURRAY, R. M. (1989) Obstetric complications and schizophrenia: methodology and mechanisms. In *Schizophrenia: Scientific Progress* (eds S. C. Schulz & C. A. Tamminga). New York: Oxford University Press.

MCGUFFIN, P., FARMER, A. & GOTTESMAN, I. I. (1987) Is there really a split in schizophrenia: the genetic evidence. *British Journal of Psychiatry*, **150**, 581–592.

MCNEIL, T. F. (1987) Perinatal influences in the development of schizophrenia. In *Biological Perspectives of Schizophrenia* (eds H. Helmchen & F. A. Henn). Bristol: John Wiley.

—— & KAIJ, L. (1978) Obstetric factors in the development of schizophrenia. In *The Nature of Schizophrenia* (eds L. C. Wynne, R. L. Cromwell & S. Matthysse). New York: John Wiley.

MURRAY, R. M. & LEWIS, S. W. (1987) Is schizophrenia a neurodevelopmental disorder? *British Medical Journal*, **295**, 681–682.

NATIONAL INSTITUTE OF MENTAL HEALTH (1976) Abnormal Involuntary Movement Scale (AIMS). In *ECDEU Assessment Manual* (ed W. Guy). Rockville: US Department of Health, Education & Welfare.

NIMGAONKAR, V. L., WESSELY, S. & MURRAY, R. M. (1988) Prevalence of familiality, obstetric complications, and structural brain damage in schizophrenic patients. *British Journal of Psychiatry*, **153**, 191–197.

O'CALLAGHAN, E., LARKIN, C., REDMOND, O., *et al* (1988) 'Early onset schizophrenia' after teenage head injury: a case report with magnetic resonance imaging. *British Journal of Psychiatry*, **153**, 394–396

——, —— & Waddington, J. L. (1990) Obstetric complications in schizophrenia and the validity of maternal recall. *Psychological Medicine*, **20**, 89–94.

Orzack, M. H. & Kornetsky, C. (1971) Environmental and familial predictors of attentional behaviour in chronic schizophrenics. *Journal of Psychiatric Research*, **9**, 21–29

Owen, M. J., Lewis, S. W. & Murray, R. M. (1988) Obstetric complications and schizophrenia: a computed tomographic study. *Psychological Medicine*, **18**, 331–339.

Parnas, J., Schulsinger, F., Teasdale, T. W., *et al* (1982) Perinatal complications and clinical outcome within the schizophrenia spectrum. *British Journal of Psychiatry*, **140**, 416–420.

Reitan, R. M. (1958) Validity of the trail making test as an indicator of organic brain dysfunction. *Perceptual and Motor Skills*, **8**, 271–276.

Schooler, N. R. & Kane, J. M. (1982) Research diagnoses for tardive dyskinesia. *Archives of General Psychiatry*, **39**, 486–487.

Waddington, J. L. (1987) Tardive dyskinesia in schizophrenia and other disorders: associations with ageing, cognitive dysfunction and structural brain pathology in relation to neuroleptic exposure. *Human Psychopharmacology*, **2**, 11–22.

—— (1988) The ageing brain, neuroleptic drugs and the enigma of schizophrenia. *Irish Journal of Medical Science*, **157**, 135–141.

—— & Youssef, H. A. (1987) Is schizophrenia a neurodevelopmental disorder? *British Medical Journal*, **295**, 997–998.

—— & —— (1988) The expression of schizophrenia, affective disorder, and vulnerability to tardive dyskinesia in an extensive pedigree. *British Journal of Psychiatry*, **153**, 376–331.

——, ——, Dolphin, C., *et al* (1987) Cognitive dysfunction, negative symptoms and tardive dyskinesia in schizophrenia: their association in relation to topography of involuntary movements and criterion of their abnormality. *Archives of General Psychiatry*, **44**, 907–912.

Walker, E. & Shaye, J. (1982) Familial schizophrenia: a predictor of neuromotor and attentional abnormalities in schizophrenia. *Archives of General Psychiatry*, **39**, 1153–1156.

Wegner, J. T., Catalano, F., Gibraltar, J., *et al* (1985) Schizophrenics with tardive dyskinesia: neuropsychological deficit and family psychopathology. *Archives of General Psychiatry*, **42**, 860–865.

Weinberger, D. R. (1987) Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry*, **44**, 660–669.

Weinhold, P., Wegner, J. T. & Kane, J. M. (1981) Familial occurrence of tardive dyskinesia. *Journal of Clinical Psychiatry*, **42**, 165–166.

Yassa, R. & Ananth, J. (1981) Familial tardive dyskinesia. *American Journal of Psychiatry*, **138**, 1618–1619.

Youssef, H. A. & Waddington, J. L. (1988*a*) Primitive (developmental) reflexes and diffuse cerebral dysfunction in schizophrenia and bipolar affective disorder: overrepresentation in patients with tardive dyskinesia. *Biological Psychiatry*, **23**, 791–796.

—— & —— (1988*b*) Involuntary orofacial movements in hospitalised patients with mental handicap or epilepsy: relationship to developmental/intellectual deficit and presence or absence of long-term exposure to neuroleptics. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 863–865.

Zec, R. F. & Weinberger, D. R. (1986) Relationship between CT scan findings and neuropsychological performance in chronic schizophrenia. *Psychiatric Clinics of North America*, **9**, 49–61.

Eadbhard O'Callaghan, MRCPsych, *Senior Registrar in Psychiatry*; Conall Larkin, MRCPsych, FRCP(C), *Consultant Psychiatrist, Cluain Mhuire Family Centre, Co. Dublin*; Anthony Kinsella, MSc, FIS, *Statistician, Dublin Institute of Technology*; *John L. Waddington, MA, MSc, PhD, *Reader in Clinical Pharmacology, Department of Clinical Pharmacology, Royal College of Surgeons in Ireland, St Stephen's Green, Dublin 2, Ireland*

*Correspondence

British Journal of Psychiatry (1990), **157**, 585–592

Predictors of Improvement in Tardive Dyskinesia Following Discontinuation of Neuroleptic Medication

WILLIAM M. GLAZER, HAL MORGENSTERN, NINA SCHOOLER,
CATHY S. BERKMAN and DANIEL C. MOORE

Forty-nine chronic psychiatric out-patients (ten were schizophrenic) with tardive dyskinesia (TD) were examined monthly for a mean of 40 weeks (range 1–59 months) after discontinuation of neuroleptic medication. Complete and persistent reversibility of TD was rare (2%), but many patients showed noticeable improvement in movements within the first year of discontinuation, which was sometimes interrupted by psychological relapse. Using three separate outcome measures and appropriate model-fitting techniques for each, we identified several predictors of improvement in TD, including an affective or schizoaffective psychiatric diagnosis, chronic (over 20 years) psychiatric illness, being employed, younger age, and increased neuroleptic dose before discontinuation. Consistent findings emerging from these analyses suggest that the type and history of psychiatric illness affect the course of TD.

The natural course of tardive dyskinesia (TD) has been extremely difficult to study because it may be influenced not only by the administration of neuroleptic medication before its development but also by these drugs administered after TD has developed. Thus, it is useful to study the course of TD in patients free of neuroleptics. Such studies have been difficult to carry out because patients with schizophrenia are rarely able to be treated clinically without neuroleptic medication for extended periods of time. Previous studies have followed the course of TD after discontinuation of neuroleptics (Uhrbrand & Faurbye, 1960; Hunter *et al*, 1964; Pryce & Edwards, 1966; Paulson, 1968; Crane *et al*, 1969; Degkwitz, 1969; Edwards, 1970; Hershon *et al*, 1972; Crane, 1973; Yagi *et al*, 1976; Quitkin *et al*, 1977; Jeste *et al*, 1979), but many of them were conducted before the use of standardised rating scales and few have used multiple measures of movement outcome. Many studies are limited because patients relapsed before adequate follow-up.

In a previous study (Glazer *et al*, 1984), we used multiple assessments to measure the outcome of TD after discontinuation of neuroleptics in 31 out-patients with and without SADS–L–RDC diagnoses of schizophrenia (Endicott & Spitzer, 1978; Spitzer *et al*, 1978). A follow-up study that included non-schizophrenic patients offered the advantage of a patient population that could go longer without neuroleptic medication and allow more opportunity to study movement outcome. In addition, such a study offered the opportunity to examine differences in outcome between schizophrenic and non-schizophrenic diagnoses. Of the discontinuation studies mentioned above, few attempted to quantify predictors of movement outcome after cessation of neuroleptic therapy (Pryce & Edwards, 1966; Hershon *et al*, 1972; Yagi *et al*, 1976; Jeste *et al*, 1979). Of these, only one (Jeste *et al*, 1979) used multivariable statistical methods. In our earlier study (Glazer *et al*, 1984) we found that complete reversibility of TD was rare but that TD improved if patients remained off neuroleptic medication. Because of the small sample size ($n = 33$) and 18-month follow-up, we were unable to identify statistically reliable predictors of outcome in TD after discontinuation of neuroleptics. In this study, we have extended our previous work by adding 16 patients, and by extending the follow-up to a maximum of five years to allow us to determine quantitatively which demographic and clinical variables predict TD outcome after neuroleptic medications are discontinued.

Method

This is a prospective study of the course of involuntary movements of patients with TD after neuroleptic medication was discontinued. Patients were selected from the Tardive Dyskinesia Clinic, a part of the out-patient division of the Connecticut Mental Health Center. The clinic has been described by Glazer & Moore (1981) and Glazer (1986). As of July 1985, when this study ended, the TD clinic had over 400 patients whose diagnoses of TD were based on clinical appearance, a six-month history of neuroleptic use, a total score on the Abnormal Involuntary Movement Scale (AIMS, Smith *et al*, 1979) of three or more, and lack of evidence of an alternative neurological diagnosis (Granacher, 1981). The total AIMS score included at least one body area with a score of two (mild) or more.

Thirty-six patients in this study did *not* meet SADS–L–RDC criteria for either schizophrenia or schizoaffective disorder, had a diagnosis of TD as defined above, and had not been admitted within the last two years. The duration of TD was determined by an assessment of the patient's verbal report, clinical records, and reports of the therapist and significant others. The SADS–L–RDC diagnoses of these patients were as follows: 14 had a major depressive disorder, one had an unspecified psychosis, three had lifetime bipolar disorder, four had chronic intermittent depression, two had personality disorder, one was alcoholic, two had generalised anxiety disorder, and one (2.8%) had Briquet's disorder. Eight of the 36 patients were not diagnosable by SADS–RDC criteria – two patients with mild mental retardation, two with dementia secondary to alcohol abuse, four patients with insufficient data from patient report or chart review – but all available data on these eight revealed no evidence of schizophrenia or schizoaffective disorder. For these patients, medication was discontinued under the supervision of the TD Clinic staff.

During the intake period, 13 additional patients with TD did not meet the above diagnostic criteria for discontinuation, but had discontinued neuroleptic medication, either because of their therapists' recommendations (eight cases) or because of their own decision (five cases). Ten of these patients met SADS–RDC criteria for schizophrenia and three (23%) were schizoaffective. These patients were included in the study as long as they could be examined within one month after discontinuation.

All baseline information was obtained by a research nurse at the time of the SADS–L interview. This information included demographic and clinical history as well as an assessment of movement status with the AIMS and psychological status with the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962). Data sources included patient reports, clinical staff reports and reviews of all available medical records. For purposes of this study, the following demographic and clinical variables were obtained and, for purposes of analysis, were categorised as follows: age (≥ 55 years v. <55 years), sex, race (white v. non-white), employment status (employed v. unemployed), living arrangement (lives alone v. lives with others), marital status (married v. not married), SADS–L–RDC diagnosis (treated as two binary variables: affective/schizoaffective v. other; or schizophrenia v. other), neuroleptic dose at start of discontinuation in chlorpromazine equivalents (CPZE) (Hollister, 1977) (dichotomised at the median: >100 mg v. ≤ 100 mg), cumulative duration of psychiatric admissions (≤ 6 months v. >6 months), duration of illness (≥ 20 years v. <20 years), duration of neuroleptic exposure (≥ 120 months v. <120 months), current use of low-potency neuroleptics (chlorpromazine and thioridazine) v. other high-potency neuroleptics, baseline total AIMS score (≥ 6 v. <6), duration of TD (≥ 1 year v. <1 year) and time to complete discontinuation of neuroleptic medication (0 weeks v. >0 weeks).

All ratings were performed by a research nurse under the supervision of the two study psychiatrists (WMG and DCM). Intraclass correlation coefficients between pairs of raters of the total AIMS and BPRS scores ranged between 0.78 and 0.94 in 32 joint inter-rater sessions during the study. Information for SADS–L–RDC diagnosis came from patient reports, staff reports and review of all available records. Following the SADS–L interview and the establishment of the RDC diagnosis, the research nurse notified the clinical staff if the diagnosis was not schizophrenia or schizoaffective disorder. Patients were gradually withdrawn from neuroleptic and anti-Parkinsonian medication over whatever time was clinically appropriate (0–16 weeks). In most cases, the day on which these medications were stopped was planned in advance and the patient was seen by the study team on that day. Involuntary movements were rated at least once before any change in neuroleptic dose and again on or near the day of medication discontinuation. The baseline TD severity score was the average of the total AIMS scores obtained at the first assessment, (i.e. before any change in neuroleptic dose), during the dose-reduction period, and at time of discontinuation. Other medications, such as antidepressants, lithium, or anti-anxiety agents, were managed clinically.

Following examination of each patient on the day of discontination, patients were evaluated monthly until either the study ended (July 1985) or they relapsed – requiring neuroleptic medication because of worsening of psychiatric symptoms – left treatment or died. During follow-up visits, the psychiatric nurse assessed medication status and performed AIMS and BPRS examinations. For purposes of the analysis, monthly ratings were collapsed into quarterly periods (i.e. mean of ratings taken over three months).

We were especially interested in how TD improves after discontinuation of neuroleptic medication. In assessing improvement, a major concern was the variability of TD movements over time in the same patients – even under relatively stable conditions, with no medication changes or shifts in psychological symptoms. We chose three complementary outcome measures that reflected different aspects of changing TD severity. Whereas an analysis of only one of these measures might have been misleading because of the variability of TD, consistent findings across the three measures would help to distinguish important effects from chance findings.

'First improvement' of TD at any follow-up visit was defined as a decrease in mean total AIMS score to at least half that at baseline, or at least a three-point decrease from baseline total AIMS score, at any follow-up visit, conditional on being kept off neuroleptics. This measure was analysed by considering the follow-up time for each patient – until either first improvement or withdrawal from observation. The second measure, *'overall improvement'*, indicates persistent improvement. A patient was classified as an overall improver if for more than half of the follow-up visits, he/she exhibited either a 50% improvement in movement severity or a decrease of three or more points in the total AIMS score. The third outcome measure is *'mean improvement'*, which measures the average degree of long-term improvement. Mean improvement is the difference between the baseline total AIMS score and the average quarterly AIMS scores after the first month of discontinuation. Thus, every patient followed for more than a month off neuroleptic medication could be assigned a change score, reflecting the amount of his/her average improvement or worsening over the total period.

Statistics

The statistical methods used to analyse these data varied according to the outcome measure under consideration. Because of relapse, loss to follow-up, or death, subjects were observed for unequal lengths of time. Analysis of data involving time to first improvement incorporated the Kaplan–Meier product-limit method to describe the average time to first improvement for the entire sample. Thus we used proportional-hazards modelling to identify predictors of first improvement. A stepwise procedure was used to select variables that were related to this outcome ($P < 0.05$). Once the 'best' model was selected, all variables that had not remained in the model were added one at a time, in separate models, to estimate the effects of each variable while controlling for the other predictors. Given the small sample size and the exploratory nature of our analysis, this last step was taken to report results of predictor variables for which the estimated effects were 'statistically non-significant'.

Mean improvement is a continuous-outcome measure that is not time dependent. Multiple linear regression analysis was used to identify predictors of this outcome measure. A stepwise selection procedure was also used for this outcome, and all variables that were not chosen by this algorithm were added individually as with the previous outcome measure.

Overall improvement is also a dichotomous measure that is not time dependent. A multiple logistic regression procedure was used to identify predictors of overall improvement. The same algorithm for selecting the best set of predictors used for the two outcomes described above was also used in these analyses. Since mean improvement and overall improvement are based on different numbers of observations per subject, we refitted both best models using a weighted procedure, where the weight was the number of observations per subject on which the outcome measure was based. We found very little difference between the weighted and unweighted results; therefore, to be consistent, all results reported in this paper are based on unweighted measures.

Results

Of the 49 patients included in this study, 23 were over 55 years old (mean 54, s.d. 13), 15 were male, 44 were white, 18 were married, 12 were employed, and 10 lived alone. These characteristics were similar to those of the larger population of neuroleptic users from which they were drawn, except for a slightly higher proportion of whites (90% v. 80%). In general, these out-patients suffered from prolonged illness with a mean neuroleptic exposure of 115 months (s.d. 68), a mean time since first diagnosis of 22 years (s.d. 11), and an estimated mean duration of TD of 10 months (s.d. 14). Twenty-eight of the patients had been in hospital for six months or less. At the time of medication change, the mean daily dose of neuroleptic medication was 255 mg CPZE (s.d. 456), and the median dose was 100 mg CPZE (range 20–3000 mg/day). This median dose was below that of the larger population of neuroleptic users from which our sample was drawn. Fourteen patients were taking a low-potency neuroleptic (chlorpromazine or thioridazine).

Of the 36 patients for whom we discontinued neuroleptic medication, the mean time to discontinuation was 4.0 weeks. The mean baseline AIMS score was 5.2 (s.d. 2.7, range 3–11). The mean follow-up for the group was 40 weeks (range 4 weeks to 59 months). Between July 1979 and July 1985 (study termination), 24 patients relapsed, 20 left treatment, one died, and four were being actively followed.

In comparing the 13 schizophrenic/schizoaffective with the 36 non-schizophrenic patients, the former had a shorter duration of neuroleptic exposure (108 v. 118 months), and a longer duration of TD (14.9 v. 9.8 months). These differences were not statistically significant ($P > 0.10$), however.

TABLE I

Point and interval estimates of the cumulative probability (P, %) of first improvement[1] at selected times after discontinuation of neurolepsis

Weeks after discontinuation	*No. of subjects at risk of first improvement at the start of the interval*	*Probability of improvement (P, %)*	*Standard error of* P *(%)*	*95% CI*
0	49	–	–	–
1	46	6.1	3.3	0, 12.8
3	37	15.1	5.3	4.8, 25.5
6	32	22.3	6.3	10.1, 33.5
9	26	27.6	6.9	14.2, 41.0
12	25	31.4	7.1	17.5, 45.3
21	21	39.1	7.8	23.8, 54.4
24	17	42.5	8.0	26.8, 58.2
30	9	62.2	8.0	46.5, 77.9
36	3	79.2	9.0	61.6, 96.8
46	1	92.8	7.0	79.1, 100.0

[1] Defined as 50% or ≥ three-point decrease in total AIMS score from baseline.

TABLE II

Estimated effects of dichotomous predictors on the rate of first improvement: results of proportional hazards analyses[1]

Predictor[1]	$\hat{\beta}$	*s.d* $(\hat{\beta})$	P	*Rate ratio*[2]	*95% C*[3]
Baseline neuroleptic dose >100 mg	1.3	0.5	0.006	3.8	1.5, 9.9
Employed	1.7	0.5	0.0003	5.7	2.1, 15.7
Affective or schizoaffective	1.2	0.5	0.01	3.3	1.3, 8.1
Age ≥55 years	−0.3	0.4	0.5	0.7	0.3, 1.7
Baseline total AIMS ≥6	−0.5	0.5	0.3	0.6	0.2, 1.5
Cumulative >6 months	0.1	0.4	0.8	1.1	0.5, 2.6
Duration of hospital stay TD ≥1 year	−0.3	0.5	0.2	0.7	0.3, 1.7
Lives alone	−0.1	0.5	0.5	1.1	0.4, 2.8
Tapered withdrawal	0.4	0.6	0.4	1.6	0.5, 4.6
Married	−0.4	0.4	0.4	0.7	0.3, 1.7
Neuroleptic exposure ≥120 months	−0.2	0.4	0.6	0.8	0.3, 1.9
Low potency neuroleptics	0.2	0.5	0.7	0.8	0.3, 2.2
White	0.7	0.8	0.3	2.1	0.5, 9.7
Schizophrenic	−0.8	0.7	0.3	0.5	0.1, 1.8
Male	−0.4	0.5	0.4	0.6	0.2, 1.7
Illness duration ≥20 years	0.3	0.5	0.5	1.4	0.5, 3.7

1. Top three predictors represent the independent effects obtained from the 'best' single model of important predictors of first improvement. Others represent the separate effects of each indicated variable, controlling for the effects of the above three.
2. Rate ratio = exp $[\hat{\beta}]$ = ratio of average rates of first improvement for two compared groups.
3. Rate ratio × exp $[\pm 1.96 \times \text{s.e.}\ (\hat{\beta})]$.

Our earliest observation (Glazer *et al*, 1984) of the absence of complete and total reversal of TD held with the addition of 16 more patients and extended follow-up time in all but one case. We did observe common improvement in TD, and the results of these analyses are presented below.

First improvement

Table I displays the probabilities of improving after discontinuation. For example, if a person could be kept off neuroleptic medication for 24 weeks, the estimated probability of showing a 50% reduction in total AIMS was 42.5% and we are 95% confident that the interval (27–58%) covers the true value, assuming no bias. By about one year, there is a 93% probability (95% CI = 79–100%) of exhibiting at least one 50% or more reduction of the baseline AIMS score. The average rate (or hazard) of first improvement during the first year of follow-up was 27/821 person-weeks = 0.033/week = 0.143/month: that is, for every 100 person-months of follow-up of TD patients during the first year after discontinuation, we would expect about 14 cases to improve.

The next question of interest was what clinical and demographic variables predict first improvement. Table II displays the 'best' proportional-hazards model as well as the results of adding each additional variable, one at a time, to this model. Three variables independently predict time until first improvement ($P<0.05$): baseline dose of neuroleptic medication >100 mg CPZE, being employed, and having a psychiatric diagnosis of schizoaffective or affective disorder. Included in Table II are the rate ratios and confidence intervals for each predictor. For example, a rate ratio of 3.3 for affective or schizoaffective diagnoses means that the average rate of first improvement was 3.3 times as great in these subjects as it was in subjects with other diagnoses, controlling for dose and employment status. Of the other predictors added to the final model, it is worth noting that patients who were white, less than 55 years old, and who were tapered off neuroleptic medication, improved faster, yet non-significantly ($P>0.3$). Furthermore, the addition of each of these variables to the best model did not change any of the results for the three significant predictors.

Mean improvement

The mean improvement for the entire sample was 0.15 points on the total AIMS score. Table III displays the results of the multiple regression analyses. There were two important predictors in the best model ($P<0.05$): baseline AIMS score and diagnosis. The estimated regression coefficients ($\hat{\beta}$) represent the average predicted difference in the reduction of the total AIMS score for the two categories of each predictor, controlling for the effects of the other predictors in the model. For example, the average improvement in TD severity was 1.9 points greater in patients with a baseline AIMS score of 6 or more than in patients with a score of less than 6 ($P=0.02$). Similarly, the average improvement was 2.5 points less in schizophrenics (schizoaffectives not included) than in non-schizophrenics ($P=0.01$), which is consistent with the finding for first improvement. In addition, controlling for baseline AIMS score and diagnoses, white, employed, unmarried patients with shorter cumulative hospital stay but longer illness showed a greater mean improvement in TD over time (yet $P>0.10$, data not shown but available upon request).

TABLE III

Estimated effects of dichotomous predictors on mean improvement of TD: results of multiple regression modelling

Predictor	β	*s.e. (β)*	*95% CI*	P
Intercept	−0.1	0.5		0.86
Baseline total AIMS ≥6	1.9	0.8	0.4, 3.5	0.02
Schizophrenic	−2.5	1.0	−4.4, −0.6	0.01

TABLE IV

Estimated effects of dichotomous predictors on the probability of overall improvement of TD: results of logistic regression modelling

Predictor	β	*s.e. (β)*	P	*Odds ratio*	*95% CI (odds ratio)*
Intercept	−2.0	0.8	0.01		
Age ≥55 years	−3.2	1.2	0.01	0.04	0.004, 0.4
Illness duration ≥20 years	2.4	1.0	0.01	10.9	1.6, 74.8

Overall improvement

Of the 49 patients followed, 10 met criteria for overall improvement. Table IV displays the results of logistic regression analysis for this outcome. The estimated effect of a predictor on overall improvement is expressed as an odds ratio. Thus, for example, the odds of overall improvement (i.e. probability of overall improvement divided by the probability of not improving) is almost 11 times greater in patients with a history of psychiatric illness of 20 or more years than it is in patients with a shorter history of illness. Similarly, the odds of overall improvement is 25 (1/0.04) times greater in patients under 55 years of age than in older patients. Of the other variables added to the best model, the strongest predictors of overall improvement included: being employed, being male, living alone, having a non-schizophrenic diagnosis, most recent neuroleptic dose exceeding 100 mg CPZE, a neuroleptic exposure duration of 120 months or more, use of chlorpromazine or thioridazine at the time of discontinuation, and a baseline AIMS score of 6 or more (yet $P>0.10$, data not shown but available upon request).

Discussion

A major finding of this study is that complete reversal of TD following neuroleptic discontinuation in chronically treated patients was rare. This pessimistic result is tempered somewhat by the fact that there was a substantial rate of improvement in patients who could be maintained off neuroleptic medication, and ten patients showed 'overall improvement'. On the other hand, a six-month risk of first improvement of less than 50% is low, as is an overall improvement rate of 10 in 49 subjects. Considering the high number of patients who relapsed, many of whom had not met SADS-L-RDC for schizophrenia or schizoaffective disorder, one has to question whether, in chronically treated patients with mixed diagnoses, the benefits of neuroleptic maintenance outweigh the risks of discontinuation. The targeted or intermittent strategy of neuroleptic maintenance (Carpenter & Heinrichs, 1983) may provide the most reasonable solution for some of these patients.

Predictors of first improvement in TD were baseline dose of neuroleptic medications >100 mg CPZE, employment, and a psychiatric diagnosis of affective or schizoaffective disorder. Predictors of greater mean improvement were baseline AIMS score of six or more and a psychiatric diagnosis other than schizophrenia. Overall improvement was predicted by a history of psychiatric illness of 20 years or more and an age of less than 55. That patients receiving neuroleptic doses over 100 mg CPZE had a higher rate of improvement, a finding similar to that of Crane *et al* (1969), we interpret as reflecting the lower absolute reduction in dose for persons receiving lower doses, resulting in less effect on TD severity. Similarly, patients with higher baseline total AIMS scores may show greater improvement after discontinuation because the higher the baseline score, the greater the opportunity to show a reduction. That age was a predictor of TD recovery was not surprising. Smith & Baldessarini (1980), who performed a *post hoc* analysis of five studies in which each patient's TD status was evaluated at least twice, one month to five years after neuroleptic discontinuation, found an improvement rate of 81% in patients younger than 54.5 years, compared with that of 46% in older patients.

The finding that employment status, psychiatric diagnosis and duration of psychiatric illness predict the outcome of TD following neuroleptic discontinuation suggests that elements of the psychiatric illness itself affect the course of TD. One of the most consistent predictors of TD improvement was employment status: compared with unemployed patients, employed patients taken off neuroleptic medication have an average first improvement rate 5.7 times greater ($P<0.001$), an overall improvement rate 4.2 times greater ($P=0.16$), and a mean reduction of TD severity 1.5 points greater ($P=0.13$). It is possible that employment status reflects some element of psychiatric illness that is not measured by such indices as duration of illness, time in hospital, medication exposure, etc. Employment may reflect mechanisms of recovery (e.g. cognitive factors) from psychiatric illness that affect or are affected by the mechanisms underlying the course of TD. Indeed, Pearlson *et al* (1984) reported an association between unemployment and enlarged lateral ventricles as measured by computerised tomography in a group of bipolar patients. The finding that patients who are employed at the time of neuroleptic discontinuation have a significantly greater rate of first improvement and tend to have both greater overall and mean improvement suggests that this measure taps a relevant dimension in prognostic factors for understanding the course of TD that has been largely overlooked.

In this study, we found that non-schizophrenic patients, the majority of whom had affective diagnoses, were 3.3 times more likely to improve than were patients with other diagnoses, especially schizophrenia. This association persists while controlling for employment status and other variables. These results provide further support for a specific relationship between affective illness and TD. Following earlier case reports of an elevated frequency of affective diagnoses among TD patients (e.g. Davis *et al*, 1976), several case-control studies (Rush *et al*, 1982; Hamra *et al*, 1983; Yassa & Schwartz, 1984) and two cross-sectional studies (Mukherjee *et al*, 1982; Yassa *et al*, 1984) lent support to this association. Several investigators (Cutler *et al*, 1981; Karson *et al*, 1985; Glazer *et al*, 1984) have found an association between movement severity and current affective state. Kane *et al* (1980, 1984) reported that patients with affective illness are more likely than other patients to develop TD within two years of first neuroleptic exposure. Glazer *et al* (1988) studied first visits to a TD clinic and found that patients with affective illness had more severe orofacial movements than did schizophrenic patients.

Thus, non-schizophrenic patients may develop TD more rapidly and more severely when exposed to neuroleptic medication than do schizophrenic patients; when the exposure is removed, their recovery is more rapid and more robust. If some forms of schizophrenia include involuntary movements that occur independently of neuroleptic exposure (Owens *et al*, 1982; Crow *et al*, 1983), these patients might account for the results obtained in this study. To address this issue, a study of a large community sample of subjects with a variety of psychiatric diagnoses unexposed to neuroleptic medication might be informative. If the above theory is correct, we would predict a higher incidence of involuntary movements in unexposed persons with schizophrenic diagnoses than in unexposed persons with other diagnoses. One could also look directly for signs of structural damage in patients whose neuroleptic medication has been discontinued. We would predict more indicators of structural damage in patients who show fewer signs of improvement in involuntary movements.

The finding that prolonged illness is positively associated with overall (long-term) improvement of TD seems puzzling. Our *a priori* hypothesis was that patients with longer illnesses would improve *less* after neuroleptic discontinuation. We expected this relationship in part because of the association of ageing and duration of neuroleptic exposure with TD. As mentioned above, we did find that older patients were less likely to show overall improvement than younger patients, but this effect was independent of illness duration. Furthermore, the effect of illness duration on overall improvement was independent of length of neuroleptic exposure and psychiatric diagnosis. It is not clear how to interpret this finding because we do not know whether it is the duration of actual psychopathology or the duration of the psychiatric diagnosis that is associated with movement outcome.

In summary, the course of TD following discontinuation of neuroleptic medication appears to be related to the type and history of psychiatric illness in our group of 49 out-patients. It is too early to know to what degree structural and/or chemical pathology inherent in the psychiatric illness or in response to neuroleptic treatment affects the occurrence and course of TD. Clearly, the prognosis of TD depends on more than neuroleptic exposure. It is possible that data pertaining to the course of TD following neuroleptic medication will provide valuable insight in the understanding of certain psychotic and affective disorders.

There are limitations of the present study that need to be recognised. We followed TD in a heterogeneous patient population for varying lengths of time after discontinuation of neuroleptic medication. We can confidently date discontinuation but we cannot date the onset of TD as reliably. Further, this study did not include a control group of patients who continued on neuroleptic medication. Several characteristics of this patient sample suggest that it is not typical of neuroleptic-treated groups in general. The majority of the sample was not schizophrenic. Of the patients who are schizophrenic, the majority chose to discontinue their own medication independently of any medical advice. The sample was made up of few blacks compared with the clinical source population from which they were selected. Finally, the doses of neuroleptic medication were relatively low. Half of the patients received a daily dose of 100 mg CPZE or less.

Another question raised by these findings is whether our definition of first improvement (i.e. 50% reduction in total AIMS score) is merely a random event, reflecting natural variability in TD severity. We doubt that this is the case for several reasons. First, it is not unusual to see responses to neuroleptic medication over time. If it takes years to develop TD, it is reasonable to expect that it may take a long time for it to improve. Second, three of the variables predicting improvement remain consistent across outcome measures. For these reasons, we conclude that first improvement is not a random event, but rather a biological response to the elimination of exposure to neurological medication.

Acknowledgements

The authors wish to thank Ann Armas for preparing the manuscript and the staff of the out-patient division of the Connecticut Mental Health Center for providing the clinical care of the patients who had participated in this study. This study was funded in part by NIMH grants MH52726 and MH30929.

References

Carpenter, W. T. & Heinrichs, D. W. (1983) Early intervention, time-limited, targeted pharmacotherapy of schizophrenia. *Schizophrenia Bulletin*, **9**, 533–542..

Crane, G. E. (1973) Persistent dyskinesia. *British Journal of Psychiatry*, **122**, 395–405.

——, Ruis, P., Kernohan, W. J., *et al* (1969) Effects of drug withdrawal on tardive dyskinesia. *Activitas Nervosa Superior*, **11**, 30–35.

Crow, T. J., Owens, D. G. C., Johnstone, E. C., *et al* (1983) Does tardive dyskinesia exist? *Moderne Probleme der Pharmakopsychiatrie*, **21**, 206–219.

Cutler, N. R., Post, R. M., Rey, A. C., *et al* (1981) Depression-dependent dyskinesias in two cases of manic-depressive illness. *New England Journal of Medicine*, **304**, 1088–1089.

Davis, K. L., Berger, P. A. & Hollister, L. E. (1976) Tardive dyskinesia and depressive illness. *Psychopharmacology Communications*, **2**, 125–130.

Degkwitz, R. (1969) Extrapyramidal motor disorders following long-term treatment with neuroleptic drugs. In *Psychotropic Drugs and Dysfunctions of the Basal Ganglia* (eds G.E. Crane & R. Gardner). Washington, DC: US Public Health Service.

Edwards, H. (1970) The significance of brain damage in persistent oral dyskinesia. *British Journal of Psychiatry*, **116**, 271–275.

Endicott, J. & Spitzer, R. L. (1978) A diagnostic interview: the schedule for affective disorders and schizophrenia *Archives of General Psychiatry*, **35**, 837–844.

Glazer, W. M. (1986) Notes from a tardive dyskinesia clinic. *Yale Psychiatric Quarterly*, **8**, 4–15.

—— & Moore, D. C. (1981) A tardive dyskinesia clinic in a mental health center. *Hospital and Community Psychiatry*, **32**, 572–574.

——, ——, Schooler, N., *et al* (1984) Tardive dyskinesia: a discontinuation study. *Archives of General Psychiatry*, **41**, 623–627

——, Morgenstern, H. & Niedzwiecki, D. (1988) Heterogeneity of tardive dyskinesia: a multivariate analysis. *British Journal of Psychiatry*, **152**, 253–259.

Granacher, R. (1981) Differential diagnosis of tardive dyskinesia: an overview. *American Journal of Psychiatry*, **138**, 1288–1297.

Hambra, B. J., Nasrallah, H., Clancy, J., *et al* (1983) Psychiatric diagnosis and risk for tardive dyskinesia. *Archives of General Psychiatry*, **40**, 347–348.

Hershon, H. I., Kennedy, P. F. & McGuire, R. J. (1972) Persistence of extrapyramidal disorders and psychiatric relapse after withdrawal of long-term phenothiazine therapy. *British Journal of Psychiatry*, **120**, 41–50.

Hollister, L. E. (1977) Anti-psychotic medications and the treatment of schizophrenia. In *Psychopharmacology. From Theory to Practice* (eds J. D Barchas, P. A Berger, R. D. Ciaranello, *et al*). New York: Oxford Publishing Press.

Hunter, R., Earl, C. J. & Thornicroft, S. (1964) An apparently irreversible syndrome of abnormal movements following phenothiazine medication. *Proceedings of the Royal Society of Medicine*, **157**, 758-762.

Jeste, D., Potkin, S., Feder, S., *et al* (1979) Tardive dyskinesia: reversible and persistent. *Archives of General Psychiatry*, **36**, 585–590.

Kane, J., Wegner, J. T., Stenzler, S., *et al* (1980) The prevalence of presumed tardive dyskinesia in psychiatric inpatients and outpatients. *Psychopharmacology*, **69**, 247–251.

——, Woerner, M., Weinhold, P., *et al* (1984) Incidence of tardive dyskinesia: five year data from a prospective study. *Psychopharmacology Bulletin*, **20**, 39–40.

Karson, C. G., Jeste, D. V. & Bigelow, L. B. (1985) Tardive dyskinesia and psychopathology in chronic schizophrenia: a cross-sectional study. *Comprehensive Psychiatry*, **26**, 388–391.

Mukherjee, S., Rosen, D. & Cardenas, C. (1982) Tardive dyskinesia in psychiatric outpatients: a study of prevalence and association with demographic, clinical and drug history variables. *Archives of General Psychiatry*, **39**, 466–469.

Overall, J. E. & Gorham, D. R. (1962) The brief psychiatric rating scale. *Psychological Reports*, **10**, 799–812.

Owens, D. G. C., Johnstone, E. C. & Frith, C. D. (1982) Spontaneous involuntary disorders of movement. *Archives of General Psychiatry*, **39**, 452–461.

Paulson, C. W. (1968) An evaluation of the permanence of the "tardive dyskinesia". *Diseases of the Nervous System*, **24**, 692–694.

Pearlson, G. D., Garbacz, D. J., Tompkins, R. H., *et al* (1984) Clinical correlates of lateral ventricular enlargement in bipolar affective disorder. *American Journal of Psychiatry*, **141**, 253–256.

Pryce, I. G. & Edwards, H. (1966) Persistent oral dyskinesia in female mental hospital patients. *British Journal of Psychiatry*, **112**, 983–987

Quitkin, F., Rifkin, A. & Gochfeld, L. (1977) Tardive dyskinesia: are first signs reversible? *American Journal of Psychiatry*, **134**, 84–87.

Rush, M., Diamond, F. & Alpert, M. (1982) Depression as a risk factor in tardive dyskinesia. *Biological Psychiatry*, **17**, 387–392.

Smith, J. M., Kucharski, L. T. & Oswald, W. T. (1979) Tardive dyskinesia: effect of age, sex and criterion level of symptomatology on prevalence estimates. *Psychopharmacology Bulletin*, **15**, 69–71.

—— & BALDESSARINI, R. J. (1980) Changes in prevalence, severity and recovery in tardive dyskinesia with age. *Archives of General Psychiatry*, 37, 1368–1373.

SPITZER, R. L., ENDICOTT, J & ROBINS, E. (1978) Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*, 35, 773–782.

UHRBRAND, L. & FAURBYE, A. (1960) Reversible and irreversible dyskinesia after treatment with perphenazine, chlorpromazine, reserpine, and electroconvulsive therapy *Psychopharmacologia*, 1, 408–418.

YAGI, G , OGITA, K., OHTSUHA, N , *et al* (1976) Persistent dyskinesia after long-term treatment with neuroleptics in Japan: its present status and clinical problems. *Keio Journal of Medicine*, 25, 27–35

YASSA, R., NAIR, V. & SCHWARTZ, G. (1984) Tardive dyskinesia and the primary psychiatric diagnosis. *Psychosomatics*, 25, 135–138.

—— & SCHWARTZ, G. (1984) Depression as a predictor in the development of tardive dyskinesia *Biological Psychiatry*, 19, 441–444.

* William M. Glazer, MD, *Associate Professor of Psychiatry, Yale University School of Medicine, Director, TD Clinic*; Hal Morgenstern, PhD, *Associate Professor of Epidemiology, UCLA School of Public Health*; Nina Schooler, PhD, *Director, Psychotic Disorders, Research Program, Department of Psychiatry, University of Pittsburgh, School of Medicine*; Cathy S. Berkman, PhD, *Associate Research Scientist, Columbia University, School of Public Health*; Daniel C. Moore, MD, *Assistant Clinical Professor of Psychiatry, Yale University School of Medicine*

*Correspondence: *School of Medicine, Department of Psychiatry, 34 Park Street, New Haven, Connecticut 06519, USA*

British Journal of Psychiatry (1990), 157, 593–597

Symptom Specificity in Patients with Panic

PAUL LELLIOTT and CHRISTOPHER BASS

Thirteen patients with panic disorder with predominantly cardiorespiratory (CR) symptoms were compared with seven patients with predominantly gastrointestinal (GI) symptoms in an experimental procedure that involved exposure to phobia talk and voluntary hyperventilation (VHV). The CR patients had not only higher baseline anxiety, but also during phobia talk had a greater fall in pCO$_2$ and reported more respiratory symptoms than the GI patients. Moreover, the CR group found VHV more unpleasant and more like their panic attacks than the GI panickers, and reported more physical symptoms after it. These findings suggest that patients with PD are not only heterogeneous with respect to the system to which panic symptoms refer (CR or GI) but that provoking arousal in one system is more likely to produce distress if that system is the major focus of complaint. These findings, if replicated, would not support the suggestion that panic disorder is a uniform illness.

The symptoms of panic according to DSM–III–R (American Psychiatric Association, 1987) are referable to a number of body systems – respiratory, cardiovascular, neurological, and gastrointestinal. Associated with the panic symptoms are characteristic catastrophic cognitions. Hibbert (1984) claims that the connection between a patient's particular cluster of somatic symptoms and accompanying cognitions are "understandable"; for instance, those who experience palpitations, chest tightness and breathlessness interpret these symptoms as a heart attack. If such specificity exists it might explain the inability of any single challenge test (lactate infusion, yohimbine, caffeine, carbon-dioxide inhalation or hyperventilation) to induce panic in all patients with panic disorder (PD) (Clark & Hemsley, 1982); quite simply, the physiological changes produced might not mimic the somatic symptoms of a panic closely enough to provoke the catastrophic cognition.

It has been known for many years that: (a) individuals tend to respond to stress with their own 'specific' pattern of autonomic activation, arousal for an individual being maximal in the same physiological function whatever the stressor (Lacey *et al*, 1953); and (b) in patients with psychosomatic complaints "the particular physiological mechanism of that complaint is specifically susceptible to activation by stressful experience" (Malmo & Shagass, 1949).

Hyperventilation causes a characteristic array of physical symptoms (Bass *et al*, 1987) and produces a greater degree of distress in panic patients than in normal controls or patients with generalised anxiety (Rapee, 1986). Many PD patients have panic symptoms referable to the cardiovascular (Klerman, 1986) and respiratory (CR) systems. Lydiard *et al* (1986) drew attention to a subgroup of patients meeting criteria for PD whose principal somatic symptoms were gastrointestinal (GI) (abdominal pain, abdominal cramping, flatulence, urge to defaecate and diarrhoea) and whose reason for avoidance was fear of being faecally incontinent. These patients provide an intriguing opportunity to test a number of hypotheses.

This study compares the subjective and physiological responses of these two groups of panic patients (with predominantly CR and predominantly GI symptoms) to a psychological stressor and voluntary hyperventilation (VHV) and poses two questions.

(a) Will the two groups respond with equal distress to VHV, which produces sensations which are more referable to the CR than GI system?

(b) Will a psychological stressor produce reports of symptoms and physiological changes maximal in the system of principal complaint (i.e. either GI or CR) in the two groups of patients with PD?

Method

Patients were drawn from consecutive referrals to the authors' units. All met DSM–III–R criteria for PD with agoraphobic avoidance confirmed by interviews with psychiatrists trained in the use of the Upjohn version of the Structured Clinical Interview for DSM–III (SCID–UP; Spitzer & Williams, 1983).

Group I (CR) consisted of 13 patients whose panic symptoms were predominantly cardiovascular and respiratory, and whose fears during panic were of impending death, heart attack, or collapse. All 13 reported at least three of the following four symptoms (listed in DSM–III–R) during one of their panic attacks: shortness of breath (dyspnoea); choking or smothering sensations; palpitations or accelerated heart rate (tachycardia); chest pain and discomfort.

Group II (GI) comprised seven patients whose panic symptoms were predominantly gastrointestinal. In addition to panic, all reported: abdominal discomfort; abdominal cramps; tenesmus; and diarrhoea, and fears of faecal incontinence during a panic attack.

The groups were of unequal size because of the small number of GI patients referred to the units. In a retrospective study (unpublished data), over ten years the GI patients comprised approximately 10% of agoraphobic referrals.

On the same day, before testing, the following ratings were made:

(a) the Fear Questionnaire (Marks & Matthews, 1979) – 15 self-rated items yielding three subscales (agoraphobia, blood-injury phobia, and social phobia), each with a range of 0–40 (higher scores denoting more anxiety and avoidance)
(b) the 90-item Symptom Checklist (SCL-90; Derogatis *et al*, 1973)
(c) a checklist of 37 self-rated symptoms clinically related to hyperventilation, for which subjects were asked if they had experienced any of them during the past week (Grossman & DeSwart, 1984).

During the experimental procedure a nasal catheter sampled end-tidal pCO_2 and measured respiratory rate; electrodes attached to ankles and wrists recorded heart rate. A box with nine buttons numbered 0 (complete calm) to 8 (panic) placed in front of the subjects allowed them to rate anxiety at a signal from the experimenter. Permanent tracings of pCO_2, respiratory rate, heart rate, and subjective anxiety were recorded on a polygraph.

Full details of the laboratory procedure are given in a separate report (Bass *et al*, 1989). Briefly, after clinical ratings subjects were taken into the laboratory, seated upright in a comfortable chair, had electrocardiograph electrodes and nasal catheter attached, and were shown how to rate subjective anxiety. Patients were acclimatised for five minutes and then told about the experiment:

> "This experiment studies how a certain bodily function behaves at rest and in response to certain tasks and instructions. I will be talking to you during the next 25 minutes and will ask you to do certain things. I want you to signify at specified times how anxious you feel by pressing a button in front of you. You will be debriefed at the end."

Details of the provocation test and the word 'panic' were omitted. The two stressors were phobia talk and VHV.

(a) *Phobia talk.* After five minutes' silence, patients were asked to imagine a phobic scene taken from the top of their hierarchy of feared situations (this scale had been determined at an earlier interview). For five minutes the experimenter described the fearful situation, emphasising stimulus propositions rather than response propositions (Lang, 1979). That is to say, images of a phobic scene such as a crowded supermarket, standing in a queue, etc., were described (stimulus content) as opposed to physical symptoms of anxiety such as trembling and incontinence (response content). This was done because emphasis on response propositions might spuriously provide evidence for specific physiological activation.
(b) *Voluntary hyperventilation (VHV).* After a further five minutes of silence, subjects were asked to overbreathe at a rate of 30–40 breaths per minute for three minutes, after which they were requested to stop overbreathing.

The two provocation tests were presented in the same order for all subjects (phobia talk followed by VHV) as it was anticipated that hyperventilation may produce changes in respiratory variables that would be slow to return to baseline.

After each provocation test subjects were asked again to complete the 37-item check-list for any symptoms experienced during the test. For technical reasons this check-list was not completed by three patients from group I after phobia talk and one patient from group I after VHV. After the phobia talk, subjects were asked to rate their clarity of visual imagery (0 (none) to 8 (total)) and after VHV all subjects completed a hedonic visual-analogue scale ranging from 0 "the most pleasant feeling ever experienced" to 10 "the most unpleasant feeling ever experienced." After VHV, subjects were also asked to rate on two five-point scales: "How many of the complaints that brought you to the doctor were reproduced?", and "How similar were the symptoms produced during the overbreathing to those you experienced in the agoraphobic/panic situation?" Higher scores denote closer similarity. The procedure and timing of measures are outlined in Table I.

TABLE I

Ratings	*Sequence of procedures: min*											
	Baseline		*Phobia talk*			*Rest*		*VHV*[1]			*Recovery*	
	1	*4*	*6*	*8*	*10*	*11*	*14*	*16*	*17*	*18*	*19*	*23*
Subjective anxiety	X	X	X	X	X	X	X	X	X	X	X	X
End-tidal pCO_2	X	X	X	X	X	X	X	X	X	X	X	X
Respiratory rate	X	X	X	X	X	X	X	X	X	X	X	X
Heart rate	X	X	X	X	X	X	X	X	X	X	X	X
Hyperventilation checklist						X						X
Similarities questionnaire												X
Hedonic scale												X

[1] Voluntary hyperventilation at 30 breaths/min.

Baseline scores were obtained at four minutes (for phobia talk) and 14 minutes (for VHV), and changes from baseline of anxiety, pCO_2, respiratory rate and pulse rate were compared using two sample t-tests. Because of the heterogeneous variance between the two groups, the questionnaire scores and data from the hedonic scale and similarities scale were analysed using the non-parametric Mann–Whitney U-test. Comparison of checklist symptoms reported by the two groups was made using Fisher's exact probability test (two-tailed).

Results

The GI patients were younger than the CR patients and had shorter duration of symptoms (but closely similar ages of onset – approximately 25 years). They were also less severly ill, with lower scores on the social phobia and agoraphobia scales of the Fear Questionnaire and certain factors of the SCL-90 (Table II).

The CR patients were more anxious than the GI group throughout the experiment (Fig. 1), but increases in anxiety from baseline were not significantly different during phobia talk or VHV. CR patients had significantly lower pCO_2 levels at rest and their pCO_2 fell significantly more than that of the GI patients during and after phobia talk (at 8, 10 min and 11 min, $P<0.05$ for all; Fig. 1). During VHV both groups achieved a similar reduction in pCO_2:15.5 mmHg for group I and 17.7 mmHg for group II. Respiratory rates were closely similar throughout the procedure, but heart-rate responses were markedly different during and after VHV. Surprisingly, the GI panickers showed significantly greater increase in heart rate at 18 minutes than the CR panickers ($P<0.01$).

TABLE II

Demographic and psychiatric data (medians and ranges) in the two groups of panic patients

	Group I Cardio-respiratory (n = 13)	*Group II Gastro-intestinal (n = 7)*
Age: years	40.0 (21–60)	29.0 (25–44)
Duration of symptoms:		
years	12.0 (2–41)*	6.0 (2–12)
SCL-90 scores		
Somatisation	17.5 (1–37)*	5.0 (3–18)
Obsessional–compulsive	11.0 (1–31)*	3.0 (1–13)
Interpersonal sensitivity	7.0 (0.28)*	1.0 (0–13)
Depression	17.0 (0–39)	8.0 (2–26)
Anxiety	22.0 (5–34)*	9.0 (3–16)
Anger–hostility	1.5 (0–11)	2.0 (0–6)
Phobic anxiety	17.0 (5–28)**	9.0 (3–16)
Paranoid ideation	1.5 (0–12)	0 (0–3)
Psychoticism	3.0 (1–18)	2.0 (1–9)
Fear questionnaire scores		
Agoraphobia	30.0 (12–34)**	16.0 (0–24)
Blood-injury phobia	16.0 (7–28)	8.0 (0–34)
Social phobia	14.0 (0–22)**	4.0 (2–12)

*$P<0.05$, **$P<0.01$ (Mann–Whitney U-test).

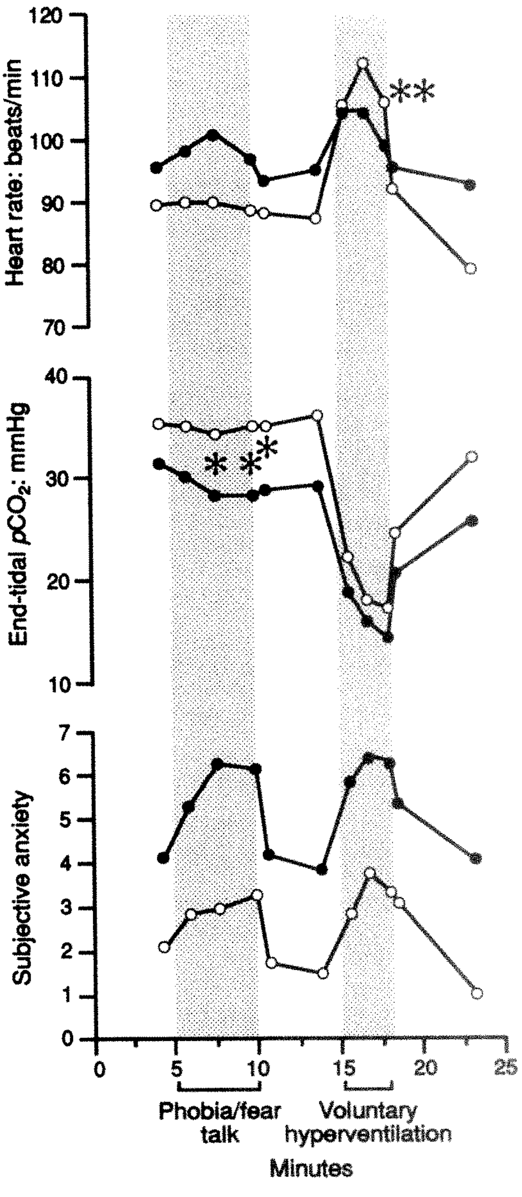


FIG. 1. Ratings of subjective anxiety, end-tidal pCO_2 and heart rate in 13 cardiorespiratory panickers (●——●) and 7 gastrointestinal panickers (○——○). Baseline scores are at 4 minutes for phobia talk and 14 minutes for VHV, and changes from baseline of anxiety, pCO_2 and heart rate were compared using two sample t-tests (*$P<0.05$, **$P<0.01$).

Panic patients in the CR group reported five of the 37 symptoms from the 37-item check-list significantly more often than those in the GI group: unable to breathe deeply enough; irregular heart beat; and tingling in fingers (all $P < 0.05$); chest pains around heart region ($P < 0.02$); and rapid heartbeat ($P < 0.01$).

During phobia talk the following somatic symptoms on the symptom check-list were reported more often by the CR patients: unable to breathe deeply enough (7/10 v. 1/7; $P < 0.05$); faster or deeper breathing than normal (8/10 v. 1/7; $P < 0.02$).

Only one of the seven GI patients reported fear of incontinence and abdominal cramps during phobia talk. This patient was anxious before the experiment and elevated baseline arousal carried over into phobia talk. Subsequently, this patient became distracted by VHV, during which she became less anxious. One other GI patient said that she may have felt physical discomfort if phobia talk had continued beyond ten minutes.

After VHV there was a trend for the CR patients to report more symptoms referable to the respiratory system than those in the GI group, viz. unable to breathe deeply enough (5/12 v. 0/7; $P = 0.1$), and need for air (4/12 v. 0/7; $P = 0.2$). None of the four gastrointestinal complaints (abdominal discomfort, cramps, tenesmus, and diarrhoea) were reported by the GI group during VHV, but one patient from both groups reported nausea (NS).

CR patients tended to find the experience of VHV more unpleasant than the GI patients (median 96 (range 52–100) compared with 65 (18–98) on the hedonic scale; $P = 0.09$), and found the experience produced sensations more similar to their normal panics (median 4.0 (range 2–5) compared with 3.0 (1–4) on the five-point similarities scale; $P < 0.05$).

Discussion

The clinical differences between the two groups of patients were unexpected. Both groups were drawn from consecutive referrals to the same hospitals, and it is unclear why those with gastrointestinal complaints should have been referred a median of 11 years earlier in their illness than those with cardiovascular/respiratory complaints. The greater duration of illness in the latter group might have explained why their illnesses were so severe – as indicated by many factors of the SCL-90 and Fear Questionnaire.

The higher levels of subjective anxiety both at rest and during provocations probably reflect this greater level of morbidity in the CR group (though some of the physiological measures were similar at rest). Differences in tonic anxiety are an almost inevitable confound in panic-induction studies and make interpretation difficult (Margraf *et al*, 1986). With this in mind, the CR patients responded to phobic talk with a greater fall in $p\text{CO}_2$ and reported more respiratory symptoms than the GI group, and the specific symptoms reported more frequently were those directly referable to respiration.

Both groups overbreathed to a similar extent when instructed to hyperventilate (as judged by fall in $p\text{CO}_2$). The CR group clearly found the experience more unpleasant, reporting greater distress on the hedonic scale and reporting more physical symptoms. As during the phobia talk, the symptoms more commonly reported during VHV were those directly related to breathing. The CR group also found the sensations produced by VHV to be more like those experienced during a panic. These findings would lend some support to a suggestion that patients with PD are not only heterogeneous with respect to the system to which panic symptoms refer (cardiovascular/respiratory, gastrointestinal, or neurological) but that provoking arousal in a system is more likely to produce distress if that system is the major focus of complaint.

This hypothesis clearly needs further testing with larger numbers of patients controlled for duration and severity of illness. If these findings are replicated and, say, panic in patients with GI symptoms is provoked by colonic distension, in those with dizziness and light-headedness by hypotensive drugs, etc., it would clearly have implications for classification, treatment and theories of aetiology of PD. It would be difficult to support the suggestion that PD is a uniform illness (as indicated by DSM–III–R) caused by a metabolic disorder (Sheehan & Sheehan, 1983) if merely provoking the feared symptoms can precipitate panics.

Acknowledgements

We thank Dr Derek Lowe for statistical advice. The data in this report were collected while Dr Lelliott was receiving a grant by the Upjohn Company.

References

American Psychiatric Association (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM–III–R). Washington, DC: APA.

Bass, C., Kartsounis, L. & Lelliott, P. (1987) Hyperventilation and its relation to anxiety and panic. *Integrative Psychiatry*, **5**, 274–291.

——, Lelliott, P. & Marks, I. M. (1989) Fear talk versus voluntary hyperventilation in agoraphobics and normals: a controlled study. *Psychological Medicine*, **19**, 669–676.

Clark, D. M. & Hemsley, D. R. (1982) The effects of hyperventilation; individual variability and its relation to personality. *Journal of Behaviour Therapy and Experimental Psychiatry*, **13**, 41–47.

Derogatis, L. R., Lipman, R. S. & Covi, L. (1973) The SCL-90: an out-patient psychiatric rating scale. *Psychopharmacology Bulletin*, **9**, 13–28.

Grossman, P. & DeSwart, J. C. G. (1984) Diagnosis of hyperventilation on the basis of reported complaints. *Journal of Psychosomatic Research*, **28**, 97–104.

Hibbert, G. A. (1984) Hyperventilation as a cause of panic attacks. *British Medical Journal*, *i*, 263–264.

Klerman, G. (1986) Current trends in clinical research on panic attacks, agoraphobia, and related anxiety disorders. *Journal of Clinical Psychiatry*, **47** (suppl. 6), 37–39.

Lacey, J. I., Bateman, D. E. & Van Lehn, R. (1953) Autonomic response specificity. *Psychosomatic Medicine*, **15**, 8–21.

Lang, P. J. (1979) A bio-informational theory of emotional imagery. *Psychophysiology*, **16**, 495–512.

LYDIARD, R. B., LARAIA, M. T., HOWELL, E. F., *et al* (1986) Can panic disorder present as irritable bowel syndrome? *Journal of Clinical Psychiatry*, **47**, 470–472.

MALMO, R. & SHAGASS, C. (1949) Physiologic study of symptom mechanisms in psychiatric patients under stress. *Psychosomatic Medicine*, 11, 25–29.

MARGRAF, J., EHLERS, A. & ROTH, W. T. (1986) Biological models of panic disorder and agoraphobia – a review. *Behaviour Research and Therapy*, **24**, 553–567.

MARKS, I. M. & MATTHEWS, A. M (1979) Brief standard self-rating for phobic patients *Behaviour Research and Therapy*, 17, 263–267.

RAPEE, R. (1986) Differential response to hyperventilation in panic disorder and generalised anxiety disorder. *Journal of Abnormal Psychology*, **95**, 24–28.

SHEEHAN, D. V. & SHEEHAN, K. H. (1983) The classification of phobic disorders. *International Journal of Psychiatric Medicine*, **12**, 243–266.

SPITZER, R. L. & WILLIAMS, J. B. W. (1983) *Structured Clinical Interview for the DSM-III (SCID)*. New York: New York State Psychiatric Institute.

Paul Lelliott, MB, BS, MRCPsych, *Consultant Psychiatrist, Greenwich District Hospital, Vanbrugh Hill, London SE10 9HE*; *Christopher Bass, MA, MD, MRCPsych, *Consultant Psychiatrist, Academic Department of Psychological Medicine, King's College Hospital, Denmark Hill, London SE5 9RS*

*Correspondence

British Journal of Psychiatry (1990), 157, 598–599

Prevalence of Huntington's Disease among UK Immigrants from the Indian Subcontinent

RAJ S. SHIWACH and RICHARD H. LINDENBAUM

Using the records of Churchill Hospital, Oxford, and those of genetics centres and other national institutions, the minimum prevalence of HD among immigrants from the Indian subcontinent was found to be almost half that found in the indigenous UK population. However, this observed prevalence was probably depressed, and therefore may not differ greatly from that estimated for European populations. All the identified cases were immigrants from Pakistan, the Punjab and Gujerat; none were from Bangladesh.

Huntington's disease (HD) has been reported worldwide (Hayden, 1981). Prevalence in white Caucasian populations is between 16 and 92 affected individuals per million. Low prevalence has been reported in Finns (5 per million; Palo *et al*, 1987), in Japanese subjects (3.8 million; Kishimoto *et al*, 1957), and in South African blacks (0.6 per million; Hayden, 1981). There have been isolated reports of HD from India, the diagnosis being based on clinical findings and, in most cases, a family history compatible with autosomal dominant transmission (Singh *et al*, 1959; Khosla & Arora, 1973). Chhuttani (1957) identified 38 affected persons among five families from north India. However, there has been no survey of the prevalence of HD in the Indian subcontinent. Indeed, many clinicians may not be aware that this disease does occur in UK immigrants from that subcontinent; such an awareness is indispensable if the families are to be given proper counselling and access to the latest techniques of molecular genetics – pre-symptom diagnosis and pregnancy diagnosis.

Method

Cases of HD were ascertained from our own records and, by telephone and written contact, from all regional and subregional genetics centres in the UK as well as from the National Hospital for Nervous Diseases and the Association to Combat Huntington's Chorea (Combat); all contacted centres responded. In addition, one of us (RSS) checked the records of the Maudsley and Bethlem Royal Hospitals. Cases were positively identified from our own records, from four of the regional genetics centres (Birmingham, Leeds, Manchester and Nottingham), and from Combat. The diagnosis in all the cases was based on clinical findings and family history.

Results

We identified 17 immigrants (14 families) from the Indian subcontinent (Pakistan, the Punjab, and Gujerat) who were affected with HD and living in this country in 1988, and a further three affected who had died in the UK during 1985–87. In this group, the age at onset of symptoms ranged from 19 years to the late 50s (median age 44 years), and clinical signs and symptoms resembled those found in English families. One patient was initially diagnosed as schizophrenic, the diagnosis later being revised to HD; one patient committed suicide.

Discussion

Studies of HD prevalence within the UK, published during the last 20 years, give rates ranging from 52–92 per million (Hayden, 1981), and the same source quotes rates of 16–70 per million for other European countries (publications since 1970), and 22–84 per million for populations of predominantly European origin overseas (USA, Canada, Australia, whites in South Africa). Lower prevalence rates were reported by earlier studies in Europe and America, perhaps because of an emphasis on studying hospital populations rather than the whole community (Hayden, 1981).

Within the UK, there are 1.26 million persons from the Indian subcontinent (Haskey, 1988). With a minimum of 17 identified HD cases, a crude estimate of prevalence within this subpopulation is therefore 13.5 per million, or 15.9 per million if the three additional cases dying between 1985 and 1987 are included.

The UK Indian population is predominantly a young one, with only 37.4% (471 000) aged over 30 years (Haskey, 1988) compared with 58% of the indigenous UK population (Shaw, 1988). We attempted a simple age-related correction as follows. In populations of European origin, 12% of HD cases present before their 30th birthday (Hayden, 1981), but the actual prevalence of HD in the population aged below 30 years will be considerably less than this. A check on HD cases known to our department, and alive in June 1988, showed eight of 104 (7.7%) to be aged under 30 years. Assuming an overall prevalence rate in the UK population of 50 cases per million, we calculated age-related prevalence rates for HD as follows: for the UK population aged below 30 years, $50 \times 7.7/100 \times 1/0.42 = 9.2$ cases per million, and for those over 30 years of age, $50 \times 92.3/100 \times 1/0.58 = 79.6$ cases per million. Applying the age-corrected prevalence rates to the Indian population in the UK, we would expect to find 7.3 cases $((1.26-0.471) \times 9.2)$ under 30 years old and 37.5 cases (0.471×79.6) over 30 years old, a total of 44.8. We actually found 17 (20) cases in this population, almost half as many as would be expected with an overall UK prevalence of 50 per million.

Emigration and immigration policies might act as selective factors in our population. Moreover, within the UK, some cases will not be diagnosed, the family history being concealed or unknown or not available (in India many die too young to manifest the disease). Furthermore, our method of ascertainment is incomplete, since a small proportion of cases will not be known to clinical geneticists, the National Hospital, or Combat. All these factors will depress the observed prevalence, and thus the gene frequency of HD at birth within the Indian immigrant population may not differ greatly from that estimated for European population groups.

Most immigrants to the UK originate either from Pakistan, Punjab and Gujerat in India, or from Bangladesh; these areas are linguistically close (Indo-European tongues), but not necessarily genetically akin. We ascertained no cases from Bangladesh, although Bangladeshis constitute 10% of the UK Indian population (Haskey, 1988). South Indians, who are linguistically distinct (Dravidian), form a very small part of the present UK population, but we do not know whether HD is rare or common in that group (Hayden, 1981). All reports of HD in India (Chhuttani, 1957; Singh *et al*, 1959; Khosla & Arora, 1973) have been from the northern states of India and from western Pakistan (now Pakistan). The ascertained UK cases also originate from this region and the adjoining area of Gujerat.

If the mutation rate in HD is very low then, depending on the place and point in time that the original mutations occurred, HD will be spread very unevenly through the populations of the world. Higher rates of mutation for HD would result in a more homogeneous distribution among different population groups. The HD gene may have been in India for a long time. Chhuttani (1957) claimed to have traced back a family pedigree from Punjab (273 members, 27 affected) for over 200 years.

No UK Indian family was large enough for a linkage study, but colleagues are storing DNA. Identification of the specific mutations in apparently unrelated families, UK and Indian, would allow resolution of the prevailing controversy regarding a single v. multiple geographical origins. Studies of haplotypes in HD (Snell *et al*, 1989; Theilmann *et al*, 1989) have detected linkage disequilibrium for two close DNA markers on chromosome 4; Theilmann *et al* (1989) found that haplotype distributions were not markedly different for UK and non-UK families (the non-UK group included two families from India, but those results were not reported separately).

Since submitting this paper we have identified a further six cases of HD among immigrants from the Indian subcontinent.

Acknowledgements

We thank our geneticist colleagues, and Combat, for their generous sharing of data, and Dr D. C. Watt for his helpful comments. At the time of this study RSS was supported by a grant from the Wellcome Trust.

References

CHHUTTANI, P. N. (1957) Huntington's chorea in India. *Journal of the Indian Medical Association*, **29**, 156–157

HASKEY, J. (1988) The ethnic minority populations of Great Britain: their size and characteristics. *Population Trends*, **54**, 29–31

HAYDEN, M. R. (1981) *Huntington's Chorea*. Berlin/New York. Springer.

KHOSLA, S. N & ARORA, B S. (1973) Huntington's chorea – a clinical study. *Journal of the Association of Physicians of India*, **21**, 247–250

KISHIMOTO, K., NAKAMURA, M. & SOTOKAWA, Y (1957) Population genetics study of Huntington's chorea in Japan *Annual Report of the Research Institute for Environmental Medicine*, **9**, 195–211

PALO, J., SOMER, H., IKONEN, E., *et al* (1987) Low prevalence of Huntington's disease in Finland. *Lancet*, *ii*, 805–806.

SHAW, C. (1988) Latest estimates of ethnic minority populations *Population Trends*, **51**, 5–8

SINGH, A , SINGH, S & JOLLY, S. S. (1959) Huntington's chorea – a report of 4 new pedigrees from Punjab *Neurology (Bombay)*, **7**, 7–8.

SNELL, R. G., LAZAROU, L. P , YOUNGMAN, S., *et al* (1989) Linkage disequilibrium in Huntington's disease: an improved localisation for the gene. *Journal of Medical Genetics*, **26**, 673–675.

THEILMANN, J., KANANI, S., SHIANG, R., *et al* (1989) Non-random association between alleles detected at D4S95 and D4S98 and the Huntington's disease gene. *Journal of Medical Genetics*, **26**, 676–681.

*Raj S. Shiwach, MB, BS, MRCPsych, *Research Senior Registrar, Department of Medical Genetics, Churchill Hospital, Oxford, and Senior Registrar in Psychiatry, Guy's & St Thomas's Hospitals Rotation*; Richard H. Lindenbaum, BM, BCh, FRCP, *Consultant, Department of Medical Genetics, Churchill Hospital, Oxford*

*Correspondence: *Department of Medical Genetics, Churchill Hospital, Headington, Oxford OX3 7LJ*

British Journal of Psychiatry (1990), 157, 600–603

Can Brain Damage Protect Against Schizophrenia?
A Case Report of Twins

SHÔN W. LEWIS, IAN HARVEY, MARIA RON, ROBIN MURRAY and ADRIANNE REVELEY

In a pair of male monozygotic twins discordant for schizophrenia, MRI revealed no abnormality in the ill proband, but extensive white-matter damage of likely congenital origin in the psychiatrically normal twin. These findings are difficult to reconcile with multifactorial models of schizophrenia, and raise the possibility that some forms of brain damage may preclude expression of the schizophrenia genotype.

That very early brain lesions might increase later risk of schizophrenia has received much attention (e.g. Murray & Lewis, 1987), growing out of a new understanding of the processes of normal and abnormal brain development, and a drive to explain the subtle non-progressive cerebral abnormalities revealed by neuroimaging in some schizophrenic patients (Murray *et al*, 1985). However, the precise nature of the putative lesions, and the mechanisms by which they might give rise to schizophrenia 20 or more years later, are not yet understood. The model of a static lesion interacting with the long-continuing processes of brain maturation is an attractive one which is used in many neurodevelopmental disorders to explain how a protean phenotype can result from an unchanging brain lesion.

The best established and probably the most important risk factor for schizophrenia is having an affected relative. However, genetic factors may not wholly be responsible since the concordance rate for schizophrenia in monozygotic (MZ) twins is only about 50%. Using the well MZ twin to control for straightforward gene effects is a powerful way to identify non-genetic risk factors. Lateral ventricular enlargement and reduced left hemisphere density, which have been shown by neuroimaging techniques in the schizophrenic twin relative to the well twin in discordant MZ pairs (e.g. Reveley *et al*, 1987), are best interpreted as the stigmata of early brain lesions that are environmentally mediated, more severe in the proband, and act as risk factors for schizophrenia. Furthermore, preliminary results from a recent magnetic resonance imaging (MRI) study in ten pairs of MZ twins discordant for DSM–III (American Psychiatric Association, 1980) schizophrenia have shown a reduced volume of left (as well as right) temporal lobe grey matter in the affected twin, as well as relative enlargement of lateral and third ventricles (Suddath *et al*, 1989).

We report here MZ twins discordant for schizophrenia in whom the neuroimaging findings offer a challenge to the simple interpretation of co-twin control data in this area, and raise the possibility that, sometimes, early brain damage might reduce, rather than increase, later risk of schizophrenia.

Case report

Abe and Ben are 38-year-old identical twins from a professional family. There is no family history of major psychiatric disorder. Monozygosity was established by physical resemblance, a questionnaire (Torgesson, 1979) and serological matching of eight blood antigens. The twins were born one week late following a toxaemic pregnancy.

Abe was delivered first by instrumental vertex delivery and weighed 3.2 kg. From infancy he had a partial left hemiparesis. He walked at 17 months and talked at 24 months. Shortly after starting school at age five years, he developed absence attacks which occurred two or three times weekly. At school he was an average scholar and made friends easily, but was often bullied; his mother attributed this to his physical disability. When aged 14 years, he developed complex partial seizures with symptoms of derealisation and *déjà vu*, usually culminating in a tonic–clonic convulsion. At age 16 years he took two small intentional overdoses of his anticonvulsants; in retrospect he viewed these as impulsive gestures in response to stresses at school. According to him and his mother, there were no other symptoms of psychiatric disorder at that time. He left school without qualifications to work as a clerk. In his late 20s the characteristics of his epilepsy changed and he suffered automatisms where for several minutes he would appear perplexed and unresponsive and pluck at his clothes; he would be densely amnesic for these episodes. An electroencephalogram (EEG) showed a right temporal spike and wave abnormality.

At follow-up he was working full time as a clerical manager and living in his own flat. He was still single and had never had a heterosexual relationship. His epilepsy was controlled with phenytoin. At interview he was friendly with a warm affect and normal social manner. There was no history of major psychiatric illness or substance abuse on administration of the Schedule for Affective Disorders and Schizophrenia (SADS-L; Endicott & Spitzer, 1978), and interview with the schizotypal section of the Schedule for Interviewing Borderlines (SIB; Baron & Gruen, 1980) revealed no abnormality. Clinical psychiatric examination showed a normal mental state. Neurological examination showed a mild left hemiparesis with a degree of hemiatrophy on the left side. He was right handed. Estimated full-scale IQ assessed with the National Adult Reading Test (NART; Nelson, 1982) was 108.

Ben was born second by breech and weighed 2.7 kg. He walked at 11 months and talked at 24 months. At school he was quiet and studious, academically bright, but with few close friends. He obtained three good 'A'-level grades and went on to read Fine Art at university. Following the ending of a six-month heterosexual relationship, he became intermittently depressed and began to abuse cannabis regularly in order to alleviate his low mood. He took LSD on one occasion. His academic work declined in standard in his third year although he received a second-class degree. He subsequently worked as a draughtsman for two years, becoming increasingly withdrawn. At age 22 years he developed ideas that he was being watched through the windows of his house. He described how an alien force had suddenly descended on his head, causing his eyes to cross. He complained of 'tremors' in his body, believed the television was referring to him, and heard a voice echo his thoughts. He was twice admitted to a psychiatric unit at the age of 23 years. On admission, he was noted to be perplexed, self-absorbed, and spent long periods gazing at his reflection in a mirror. He giggled inappropriately and described how his bodily movements were controlled by an external force and how thoughts were inserted into his mind by 'ghosts'. An EEG showed equivocal bilateral slow wave activity. He made a partial recovery, although continuing to complain of voices conversing about him.

He never returned to work and spent most of the following 15 years in a psychiatric hospital. He experienced persistent auditory hallucination, poor self-care and believed that blood clots had formed in his arms and that his body had increased in size. These changes he attributed to "a man who lives in my brain".

At follow-up, he had spent the previous two years continuously in hospital, despite high doses of oral and depot antipsychotic medication and concerted attempts at rehabilitation. Psychiatric examination showed a gaunt, dishevelled man who had an orofacial dyskinesia and shrugged his shoulders in a tic-like movement. He was friendly and co-operative; his speech was spontaneous but showed occasional paraphasias and tangential associations. His affect was not grossly blunted. He described distressing auditory hallucinations and thought interference. His insight was partial; he admitted to being ill but was convinced of the reality of some of his abnormal experiences. Apart from the tardive dyskinesia, neurological examination was normal. There was no history of epilepsy. He was right handed. His estimated pre-morbid full-scale IQ on the NART was 120. ICD-9 (World Health Organization, 1978) and DSM-III-R (American Psychiatric Association, 1987) diagnoses were of chronic paranoid schizophrenia.

Each twin consented to undergo MRI. Both examinations were performed within three weeks of each other using the same Picker 0.5-tesla system. Contiguous 5 mm thick transverse and coronal slices were obtained with a 256 × 256 matrix using a transverse spin-echo ($T_e = 70$ ms, $T_r = 2400$ ms; 24 slices) and a coronal inversion-recovery scan ($T_e = 40$ ms, $T_i = 150$ ms, $T_r = 4420$ ms; 20 slices).

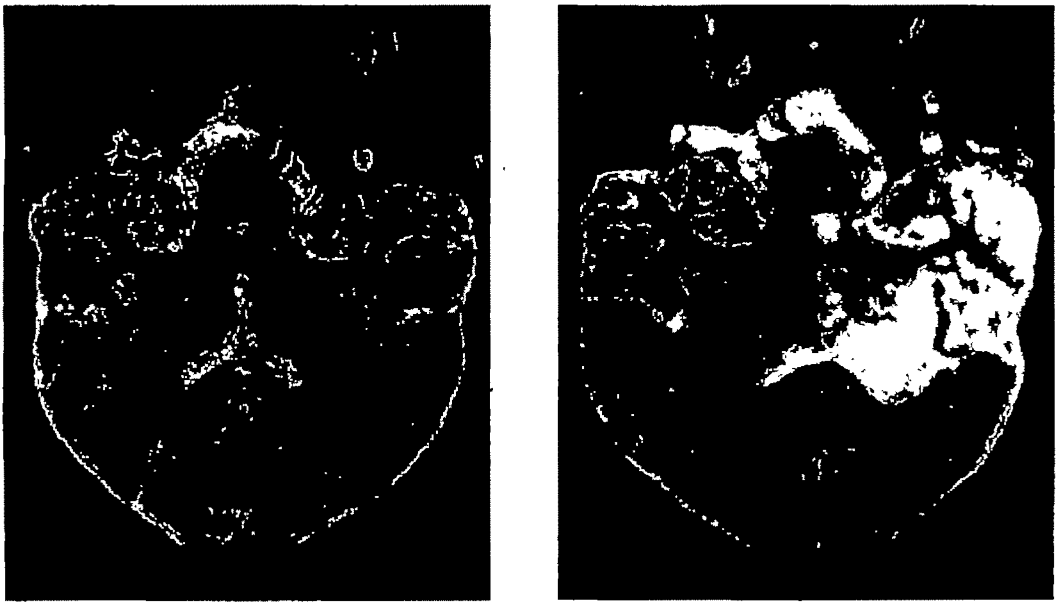

(a) (b)

FIG. 1. Coronal spin-echo MRI images through the midtemporal region in the schizophrenic twin (a) showing no abnormality, while the non-schizophrenic co-twin (b) shows extensive lesions in the right temporal lobe with consequent dilatation of the right lateral ventricle

In Abe, the non-schizophrenic twin, the scan revealed widespread areas of increased signal in the right temporal lobe, extending into the frontal and parietal area. This affected predominantly white matter but also the frontal and temporal cortex. There was marked compensatory dilatation of the right lateral ventricle and temporal horn. The right cerebral hemisphere was reduced in size compared to the left (Fig. 1). In Ben, the schizophrenic twin, no abnormality was seen on the scan (Fig. 1).

Discussion

Magnetic resonance imaging is superior to other methods of structural imaging such as computerised tomography in demonstrating white-matter lesions clearly. In the case reported here, MRI showed asymmetrical ventricular dilatation in the non-schizophrenic twin and the pathology underlying this: an extensive right-sided subcortical lesion. The lesion was associated with an apparent hypoplasia of the right hemisphere generally. This, with the history of a partial left hemiparesis evident in early infancy, points to an early developmental insult. In contrast, no abnormality was shown in the schizophrenic twin.

The abnormal exo- and endophenotypes in the non-schizophrenic twin, as well as the neurological disorder and a non-progressive brain lesion, arose from an early environmental insult, probably *in utero*. The incidence of a variety of neurodevelopmental abnormalities, including cerebral palsy and complex partial epilepsy in this case, is considerably increased in twins compared with singletons, and in MZ twins compared with DZ twins (Volpe, 1987). Most MZ foetuses share a single chorionic blood supply, which can give rise to pre-natal haemodynamic problems such as shunting of arterial blood to the detriment of one or other twin. This is the usual cause of unexpectedly discrepant birthweights in MZ twins, differences which have been shown to predict later neurological deficits as well as more subtle yet persistent functional impairments such as slightly lowered IQ scores in one twin (Munsinger, 1977). Interestingly, the interaction of an early, static cerebral lesion with post-natal development of the central nervous system (CNS) can be seen in the changing expression of the epilepsy in the psychiatrically well twin: absence attacks emerging in childhood, progressing to the psychic experiences of complex partial seizures in adolescence, and finally the appearance of automatisms in early adulthood.

This case does not fit easily with multifactorial models of schizophrenia in which the phenotype results from the sum of genetic diathesis and non-specific adverse environmental factors, since the evidence available is that the latter were more severe in the co-twin. There are two other possible explanations. One is that each twin has incurred separate environmentally-mediated insults which were sufficiently different to bring about two radically different 'phenocopies'; one exclusively neurological, the other exclusively psychiatric. Although a negative MRI scan does not rule out minor pathological changes, this hypothesis obliges us to invoke a mechanism for which there is no direct evidence, radiological or otherwise, in this particlar case and no firm experimental evidence in general (Lewis, 1989). The second explanation supposes that each member of the MZ pair carries a genotype sufficient for the expression of schizophrenia, but in the co-twin's case this expression has somehow been prevented by the brain damage. The specific evidence available tends to support this formulation and, in that it is the more parsimonious, Occam's razor would direct us to consider it the more likely. In the absence of further information, however, it is also conceivable that the history of cannabis abuse in the proband might have played a causal role, although the evidence that cannabis can *de novo* give rise to schizophrenia is inconclusive (e.g. Negrete *et al*, 1989).

There are ample precedents for the interpretation of twin discordance for a partly genetic disorder in terms of protective factors in the co-twin rather than precipitating factors in the proband, although such protective factors are more usually behavioural than organic: regular physical exercise protecting one twin against ischaemic heart disease, for instance. Whatever, this case signals caution in the uncritical use of the co-twin control method in schizophrenia. Also, the possibility that some patterns of early brain damage might actually reduce, rather than increase, the later risk of schizophrenia, deserves further exploration.

Acknowledgements

We thank the twins and the consultants involved clinically, Professor I. MacDonald and Mr D. MacManus at the Institute of Neurology, Queen Square, the Multiple Sclerosis Society, and the Medical Research Council for support.

References

AMERICAN PSYCHIATRIC ASSOCIATION (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

—— (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn, revised) (DSM-III-R) Washington, DC: APA

BARON, M & GRUEN, R. (1980) *The Schedule for Interviewing Borderlines (SIB)*. New York: New York State Psychiatric Institute.

ENDICOTT, J. & SPITZER, R. L. (1978) A diagnostic instrument: the Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, **35**, 837

LEWIS, S. W. (1989) Congenital risk factors for schizophrenia. *Psychological Medicine*, **19**, 5–13.

MUNSINGER, H. (1977) The identical-twin transfusion syndrome: a source of error in estimating IQ resemblance and heritability. *Annals of Human Genetics*, **40**, 307–321.

MURRAY, R. M. & LEWIS, S. W. (1987) Is schizophrenia a neurodevelopmental disorder? *British Medical Journal*, **295**, 681–682.

——, —— & REVELEY, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, *i*, 1023–1026.

NEGRETE, J. C. (1989) Cannabis and schizophrenia. *British Journal of Addiction*, **84**, 349–351.

NELSON, H. E. (1982) *Manual for the National Adult Reading Test*. Windsor, England: NFER Nelson Publishing.

REVELEY, M. A., REVELEY, A. M. & BALDY, R. (1987) Left cerebral hypodensity in discordant schizophrenic twins. A controlled study. *Archives of General Psychiatry*, **44**, 625–632.

SUDDATH, R. L., CHRISTISON, G., TORREY, E. F., *et al* (1989) Quantitative magnetic resonance imaging in twin pairs discordant for schizophrenia. *Schizophrenia Research*, **2**, 129.

TORGESEN, S. (1979) The determination of twin zygosity by means of a mailed questionnaire. *Acta Geneticae Medicae et Gemellologicae*, **28**, 225–236.

VOLPE, J. J. (1987) *Neurology of the Newborn*. Philadelphia: W. B. Saunders.

WORLD HEALTH ORGANIZATION (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

*Shôn W. Lewis, BSc, MPhil, MRCPsych, *Senior Lecturer, Charing Cross and Westminster Medical School, London W6 8RP*; Ian Harvey, MSc, MRCPsych, *Research Worker, Institute of Psychiatry, London SE5 8AF*; Maria Ron, PhD, MRCPsych, *Consultant Psychiatrist, National Hospitals for Nervous Diseases, London WC1*; Robin Murray, MD, FRCP, FRCPsych, *Dean, Institute of Psychiatry, London SE5 8AF*; Adrianne Reveley, MRCPsych, *Consultant Psychiatrist, Maudsley Hospital, London SE5 8AF*

*Correspondence

British Journal of Psychiatry (1990), 157, 603–605

Claimed Simulation of Insanity

A Coping Strategy in Mania

T. R. SURESH and T. N. SRINIVASAN

The coping strategy of claimed simulation of insanity observed at the onset of psychotic episodes in a manic patient is reported here. There is a need for systematic research on coping strategies in affective psychosis in order to develop techniques to help contain or prevent relapses.

Research into coping strategies is increasingly interested in the ways in which people with serious life crises or handicaps learn to deal with them consciously and purposefully (Breier & Strauss, 1983). Until recently, coping strategies adopted by patients experiencing an episode of psychosis have not received much attention, but they are currently being systematically studied, focusing especially on how they can be applied in the management of the illness. Subjective perception of prodromal changes in vegetative and emotional functions have been observed in schizophrenic patients by Herz & Melville (1980), Heinrichs *et al* (1985), Brenner *et al* (1987), Häfner & Thurm (1987) and, in an Indian population, by Shuba Kumar *et al* (1989). These studies reported that a large proportion of their patients consciously adopted different coping strategies like internal dialogue, seeking a comforting relationship, social withdrawal, seeking treatment and self-adjustment of medication in an effort to ward off an impending relapse or exacerbation of the psychosis.

Research into coping mechanisms has frequently concentrated on schizophrenic patients, those with psychoses like mania receiving little attention. Indeed, we could not find any literature dealing specifically with the issue of coping in mania. This prompted us to report the case of a man with manic psychosis who adopted the coping strategy of claiming simulation of insanity at the onset of both the psychotic episodes he experienced.

Case report

Mr S, a 26-year-old man, was admitted for treatment after a four-day period of insomnia, overactivity, irritability and excessive spending of money. The illness was preceded by a quarrel with his brothers over an issue considered trivial by them. After this quarrel the patient warned his uncle, a confidant, that he was going to act mad from then onwards so that he could keep away from his brothers. His disturbed behaviour subsequent to this warning was believed by the family to be only an 'act'. When the patient's behaviour worsened and became uncontrollable after two days, professional help was sought. A diagnosis of manic disorder was made according to DSM-III criteria (American Psychiatric Association, 1980). The episode remitted in two weeks with the use of lithium carbonate and haloperidol. After discharge from the hospital, the patient was continued on lithium carbonate (serum lithium = 30.8 mEq/l). However, he did not attend regularly for follow-up sessions and was readmitted about a year later under similar circumstances – a claim that he was going to act mad, subsequent to a family argument over a petty issue, was followed by the development of features of a manic syndrome. During this episode, the family members did not believe the patient's assertion that he was acting mad, but took the claim itself as an indicator of another episode of illness and sought professional help the day after onset. Thus, they avoided much of the distress experienced during the initial episode. The patient responded well to the same treatment as followed previously and, at one year of follow-up, he was euthymic.

Discussion

Dissimulation or 'playing down' the symptoms of an illness, considered by Slater & Roth (1977) to be a coping strategy, has been commonly observed in schizophrenics in clinical settings (Breier & Strauss, 1983). Slater & Roth (1977) point out that this dissimulation may take many interesting forms like the one described by Bleuler in which the patient insisted that he had only simulated the illness and that he was not actually ill. Jung stressed that such confessions of simulation by psychotic patients should be received with caution (Hay, 1933). Slater & Roth (1977) cite the classical, albeit controversial, example of Rudolf Hess, the Nazi war criminal, who, by this form of unusual behaviour, apparently prevented an array of international experts from reaching the obvious diagnosis. The phenomenon of dissimulation in the form of claimed simulation of insanity has been commonly observed during or after recovery from the illness. The reported case is thus of interest as the coping behaviour was the harbinger of each oncoming episode of psychosis.

Literature sometimes offers deeper insights than textbooks into the inner experiences of persons suffering from mental disturbances. With regard to coping strategies in psychotic illness a good example of this can be found in how Shakespeare's Hamlet attempted to deal with impending insanity by warning Horatio and Marcellus that he may have future occasion to act insane ("put an antic disposition on"; Bynum & Neve, 1985). This strategy was similar to the one observed in our patient, although it should be noted that the diagnosis of Hamlet's malady would probably have been depression.

The practical implications of understanding coping mechanisms in functional psychoses like mania, lie in their relevance to planning strategies for early detection, treatment and, possibly, prevention of a relapse of the illness. Chapman (1966) observed that self-monitoring of pre-psychotic affective signals appeared particularly helpful in alerting the patients to the ensuing psychotic decompensation. Such an early warning system could lead them to adopt necessary coping strategies. Although no such 'affective signals' were reported in our patient, it was possible that the patient sensed the psychological disruption occurring within him and, more or less consciously, attempted to cope with this by asserting that his future behaviour was going to be only an 'act'. This mechanism, by helping the patient falsely believe in his own assertion, seemed to have protected him, although only temporarily, from the distressing experience of a psychotic breakdown. Such coping behaviours would not only help the subject to take some ameliorative measures but could also alert the members of his or her social network to deal with the patient. This was illustrated in our report in which, when the patient made a claim of simulated psychosis for the second time, the family members, learning from a similar experience during the initial episode, made no delay in seeking psychiatric intervention. Any management plan utilising coping strategies should take into consideration the personal coping style of the given patient who, as in our case, might adopt the same or a similar behaviour every time he is in a crisis.

Conclusion

Systematic research on coping strategies of patients with affective psychoses needs to be carried out, focusing particularly on developing methods to help contain or prevent relapses.

References

American Psychiatric Association (1980) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn) (DSM-III). Washington, DC: APA.

Breier, A. & Strauss, J. S. (1983) Self-control in psychotic disorders. *Archives of General Psychiatry*, **40**, 1141–1145.

Brenner, H. D , Boker, W , Muller, J., *et al* (1987) On autoprotective efforts of schizophrenics, neurotics and controls *Acta Psychiatrica Scandinavica*, **75**, 405–414.

Bynum, W. F. & Neve, M. (1985) Hamlet on the couch. In *Anatomy of Madness. Volume I. People and Ideas* (eds W F Bynum, R. Porter & M. Shepherd), pp. 289–304. London: Tavistock.

Chapman, J. (1966) Early symptoms of schizophrenia. *British Journal of Psychiatry*, **112**, 225–251

Hafner, H & Thurm, I. (1987) Perceived vulnerability, relapse risk and coping in schizophrenia. *European Archives of Psychiatry and Neurological Sciences*, **237**, 46–53.

Hay, G. G. (1983) Feigned psychosis – a review of simulation of mental illness. *British Journal of Psychiatry*, **143**, 8–10

Heinrichs, D. W., Cohen, B. P. & Carpenter, W. T. (1985) Early insight and the management of schizophrenic decompensation. *Journal of Nervous and Mental Disorders*, **173**, 133–138.

Herz, M. & Melville, C. (1980) Relapse in schizophrenia *American Journal of Psychiatry*, **137**, 801–805.

Shuba Kumar, Thara, R. & Rajkumar, S. (1989) Coping with symptoms of relapse in schizophrenia. *European Archives of Psychiatry and Neurological Sciences* (in press).

Slater, E. & Roth, M. (1977) *Clinical Psychiatry*, p. 322. London: Baillière Tindall.

*T. R. Suresh, MBBS, DPM, *Tutor in Psychiatry, Department of Psychiatry, Sri Ramachandra Medical College, Porur, Madras 600 116, India*; T. N. Srinivasan, MD, *Assistant Professor of Psychiatry, Department of Psychiatry, Porur*

*Correspondence

British Journal of Psychiatry (1990), **157**, 605–608

Reflections of the Self: Atypical Misidentification and Delusional Syndromes in Two Patients with Alzheimer's Disease

SUSAN E. MOLCHAN, RICK A. MARTINEZ, BRIAN A. LAWLOR,
JORDAN H. GRAFMAN and TREY SUNDERLAND

Two patients with moderately severe AD, when asked directly, could identify their own images in a mirror, but also consistently misidentified their own reflections as that of another person. Both patients were paranoid and mildy depressed at times, but had no evidence of other concurrent psychotic symptoms. It appeared that mood substantially modified the nature of the symptom and the patients' reaction to it over time. These cases illustrate the ability of an organic symptom to be modified by a concurrent affective state, indicating the importance of the interaction between biological and psychological factors in the expression of such symptoms.

Psychotic symptoms are estimated to occur in 15–75% of patients with Alzheimer's disease (AD; Drevets & Rubin, 1989). Hallucinations and delusions are the most common of these symptoms, but misidentification syndromes such as Capgras' syndrome have also been reported (Drevets & Rubin, 1989). Other misidentification syndromes such as Frégoli syndrome (the belief that some familiar person has assumed the psychological identity of another, although the two are not physically alike) and the syndrome of subjective doubles have been reported primarily in patients with schizophrenia, but also in patients with organic brain disease (Joseph, 1986; Signer, 1987). It is not uncommon for AD patients to misidentify their own reflection in the mirror. Cases have also been described where patients may converse with the image in the mirror, and are unable to recognise it as their own (Rubin *et al*, 1988). Our cases are different, as the patients were able to identify their own reflections but yet frequently perceived in the reflection another person with a separate psychological identity from themselves. Our cases are also different from those where the patient recognises the image as that of the self

---

SEM is a US Government employee, and as such this material is in the public domain and cannot be copyrighted.

but believes that the image is an imposter or a double (Gluckman, 1968).

Case reports

Case 1

HT is a 61-year-old married white woman who was diagnosed as having probable AD three years ago, after a one-year history of gradual but progressive memory loss and after other causes of her symptoms had been ruled out. During much of her illness, she exhibited prominent depressive symptoms, primarily feelings of hopelessness and helplessness, sad mood, tearfulness, anxiety, and suicidal ideation. The patient had a long history of low self-esteem and of difficulty asserting herself with her husband, who was described by staff as emotionally distant and controlling. While at home alone one day, the patient ran away, explaining that she had been chased out by 'the woman', who was described as being hostile towards the patient. The husband reported that the patient identified 'the woman' when she saw her own reflection in a mirror or window.

On admission to the National Institutes of Health (NIH) clinical centre, the patient was mildly anxious and depressed. Her speech was coherent and logical, although it contained paraphasic errors and revealed word-finding difficulty. She had no evidence of hallucinations, thought disorder, or paranoid ideation other than that related to 'the woman'. The patient denied any suicidal ideation. Her abilities to abstract and to perform verbal reasoning skills were poor, as evidenced by her word fluency, sequencing skills, and ability to calculate. Her ability to draw a clock was also poor, and perseveration was evident on some of the verbal tests. She scored 13 on the Mini-Mental State Examination (MMSE; Folstein *et al*, 1975), and her Wechsler Memory Scale (WMS; Wechsler, 1945) score was 59, indicating moderately severe impairment.

The patient complained that 'the woman' had followed her to the hospital, and she wondered why 'the woman' was not at home 'cleaning the toilets' which was her only responsibility. She could point to her own image in the mirror and recognise it as herself, but would say that 'the woman', an entity separate from herself, was also present. When standing in front of the mirror, either alone or with another one or two people, she would consistently count one more person in the reflection than was actually there. She would become very upset and tearful upon seeing 'the woman' and was sometimes found hiding from her. She denied seeing any doubles of other people. When questioned, she would occasionally affirm that she believed the woman wanted to replace her, although usually she denied having that belief.

On admission to the NIH clinical centre, physical examination of the patient showed nothing abnormal, except for the presence of frontal release signs. There was no evidence of agnosia, apraxia, neglect, or prosopagnosia. A neuro-ophthalmological examination revealed no visual-field or other deficits.

After several weeks in the hospital, the patient mentioned 'the woman' less frequently and no longer seemed to be distressed by the image. When asked, she acknowledged that she continued to see 'the woman' who still spoke to her. The patient said that her interactions with 'the woman' had become friendlier and were no longer hostile. Although the patient did not meet criteria for major depression on admission, she did have some depressive symptoms that improved with the structured activities, social contacts, and supportive staff available on the unit. Depression ratings, which were done by a consensus of staff members, reflected the change in mood. Her ratings on the Dementia Mood Assessment Scale (DMAS; Sunderland *et al*, 1988) decreased from 29 to 19. On the depression item of the Bunney–Hamburg Global Rating Scale (Bunney & Hamburg, 1963), her score decreased from seven to three, a clinically significant decrease.

Case 2

LB, a 55-year-old black man, was noted by his family to become socially withdrawn and suspicious, to have difficulty handling money, and to get lost on occasion. These symptoms had become progressively worse over a period of 18 months. His past medical history was significant for alcohol abuse and amateur boxing. His mother and a paternal aunt had been diagnosed with probable AD. After an evaluation to eliminate other causes of dementia, he was diagnosed as having AD. During an out-patient follow-up visit, his caretaker mentioned that when he looked into a mirror, he sometimes did not seem to recognise the image as himself. Sometimes he became angry and accusatory towards the image. At other times, he was reported to talk with the image and laugh. When examined in the clinic, the patient was able to recognise his own reflection, but at times would also identify 'a man' who the patient said did not particularly resemble himself. He did not feel that this 'man' was trying to replace him or anyone else, and denied seeing any other such entities. His caretaker related that at times he misinterpreted shadows as 'the man', but had no other illusions, misidentifications, or hallucinations. Physical examination was normal, and there was no evidence of apraxia, agnosia, neglect, prosopagnosia, or agraphaesthesia.

The patient exhibited a full range of affect that was congruent with good mood. His speech was coherent and logical, although he had occasional word-finding difficulty. He was mildly suspicious, but otherwise had no evidence of thought disorder, hallucinations, or delusions except those that related to 'the man'. Calculations were poor, as was performance on a block design task. His ability to perform abstract reasoning was impaired. Verbal fluency was much less than normal. He scored 12 on the MMSE and 59 on the WMS, indicating moderately severe memory impairment. His most recent scores on objective ratings of depression were 17 on the DMAS and two on the Bunney–Hamburg Global Rating Scale, suggesting little baseline depression.

The symptom described above has persisted for eight months of follow-up in this patient. Fluctuations in mood, from euthymia to mild depression, were noted by staff during his in-patient stay, and these fluctuations continued during out-patient follow-up. Since he was an out-patient, we could not reliably determine whether there was a relationship between his mood and his reaction to the perceived man in the mirror. The patient also fluctuated from being mildly suspicious to frankly paranoid. When most paranoid, he would often not discuss his symptom, or would deny what he seemed to be perceiving.

Discussion

Aetiologically, misidentification syndromes were initially thought to be purely functional. In recent years, however, these syndromes have been associated with an increasing number of organic pathologies. In many of these cases, damage to the brain is bifrontal, with additional injury in the right cerebral hemisphere or temporal lobe (Christodoulou, 1977; Alexander *et al*, 1979; Morrison & Tarter, 1984; Joseph, 1986). In AD, neuropathological changes occur commonly in the frontal and temporal cortices, so a similar mechanism could possibly be at work (Drevets & Rubin, 1989). Both our patients exhibited deficits predominantly in the frontal and temporal lobes, with some right parietal lobe involvement in the first case. 'Disconnection' mechanisms have been hypothesised as a cause of such misidentification and reduplication syndromes, with disconnection of the memory and affective functions in the limbic system and temporal lobes from the interpretative and judgemental functions of the frontal lobes (Joseph, 1986).

Psychodynamic implications of other misidentification syndromes are often hypothesised to be caused by feelings of ambivalence in some significant relationship, and by splitting, as used in the Kleinian sense, to preserve the important good aspects of the person in question, while creating a vehicle (the imposter or double) on which to attribute and react to the more negative features (Gluckman, 1968; Berson, 1983). In the first case, for example, this non-assertive woman with low self-esteem, more dependent than ever because of her dementia on her overcritical, emotionally distant husband, could conceivably have displaced the hostility and anger that she felt for her husband and her situation, and directed these negative feelings, which were too threatening for her to express overtly, towards this 'other woman'. She can then blame 'the woman', rather than her husband, for causing her distress. In this case, dynamic factors may have then influenced the patient's perception of the (self) image and her reaction to it.

These cases illustrate two important points in the management of patients with similar symptoms. Firstly, although an organic aetiology for the symptoms is evident, such symptoms can be modified by the mood of the patient. In our first case, the patient's mood improved with structure and support. Conceivably, if such a patient continued to be dysthymic, antidepressant medication would have been helpful. Secondly, although initially a very distressing symptom to both patients and caretakers, the delusional system can be managed effectively by reassuring those involved, by recommending increased structure and support for the patient, and (as caretakers had already started to do) removing the initiating stimuli for the misidentification and delusion (i.e. covering up the mirrors). In these two cases, the staff were able to manage the patients effectively without the use of neuroleptics. This treatment approach is especially important in elderly, organically impaired patients who are at high risk for tardive dyskinesia.

Of primary interest is the parallel in the first case between mood and the patient's reaction to the misidentified self-image. The patient's perception of 'the woman', initially a frightening, hostile entity to her when she was relatively isolated, often criticised by her husband and depressed at home, changed to include more friendly or neutral feelings after being in a more stimulating, supportive environment in the hospital for a few weeks, during which the patient was also more consistently euthymic. In the second case, the patient also had reactions that were both friendly and hostile to the perceived image in the mirror. These reactions probably fluctuated with his mood and level of paranoia. While these changes in mood and organic symptoms may be independent, the temporal relationships suggest a connection. This modifiability of an organic symptom by affective state, which was itself modified by psychosocial intervention, is a natural example of interplay between the biological and psychological realms.

References

Alexander, M. P., Stuss, D. T. & Benson, D F. (1979) Capgras syndrome: a reduplicative phenomenon. *Neurology*, **29**, 334–339.

Berson, R. J. (1983) Capgras' syndrome. *American Journal of Psychiatry*, **140**, 969–978.

Bunney, W. E & Hamburg, D. A. (1963) Methods for reliable longitudinal observation of behavior. *Archives of General Psychiatry*, **9**, 280–294.

Christodoulou, G. N. (1977) The syndrome of Capgras. *British Journal of Psychiatry*, **130**, 556–564

Drevets, W C. & Rubin, E. H. (1989) Psychotic symptoms and the longitudinal course of senile dementia of the Alzheimer type *Biological Psychiatry*, **25**, 39–48.

Folstein, M F., Folstein, S. E & McHugh, P. R. (1975) Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatry Research*, **12**, 189–198.

Gluckman, L K. (1968) A case of Capgras syndrome. *Australian and New Zealand Journal of Psychiatry*, **2**, 39–43.

Joseph, A. B. (1986) Focal central nervous system abnormalities in patients with misidentification syndromes. *Bibliotheca Psychiatrica*, **164**, 68–79.

Morrison, R. L & Tarter, R. E. (1984) Neuropsychological findings relating to Capgras syndrome. *Biological Psychiatry*, **19**, 1119–1128.

Rubin, E. H, Drevets, W. C. & Burke, W. J. (1988) The nature of psychotic symptoms in senile dementia of the Alzheimer type. *Journal of Geriatric Psychiatry and Neurology*, **1**, 16–20.

Signer, S. F. (1987) Capgras' syndrome: the delusion of substitution. *Journal of Clinical Psychiatry*, **48**, 147–150.

Sunderland, T., Alterman, I. S., Yount, D., *et al* (1988) A new scale for the assessment of depressed mood in demented patients. *American Journal of Psychiatry*, **145**, 955–959.

Wechsler, D. (1945) Standardized memory scale for clinical use. *Journal of Psychology*, **19**, 87–95.

*Susan E. Molchan, SCN, LCS, MD, *Staff Psychiatrist, Geriatric Psychopharmacology, Laboratory of Clinical Science, Unit of National Institute of Mental Health, Bethesda, Maryland, USA*; Rick A. Martinez, MD, *Staff Psychiatrist, Unit of Geriatric Psychopharmacology, Bethesda*; Brian A. Lawlor, MD, *Assistant Professor, Department of Psychiatry, Mount Sinai School of Medicine, New York*; Jordan H. Grafman, PhD, *Chief, Cognitive Neuroscience Unit, Medical Neurology Branch, National Institute of Mental Health, Bethesda*; Trey Sunderland, MD, *Chief, Unit of Geriatric Psychopharmacology*

*Correspondence: *NIH Clinical Health Centre, 10-3D41, 9000 Rockville Pike, Bethesda, MD 20892, USA*

British Journal of Psychiatry (1990), **157**, 608–610

Delusional Misidentification, AIDS and the Right Hemisphere

PAUL CRICHTON and SHÔN LEWIS

A patient with AIDS developed the Capgras' syndrome as part of an acute confusional state resulting from an opportunistic infection. Neuropsychological testing suggested non-dominant hemisphere dysfunction with impaired facial recognition. Serial CT scanning showed a right parietal lesion which resolved after the psychosis improved. These findings provide further evidence for the hypothesis that selective visuospatial deficits underlie the development of Capgras' syndrome.

Capgras' syndrome (Capgras & Reboul-Lachaux, 1923) is characterised by the delusion that persons familiar to the patient have been replaced by physically identical imposters. The rarity of the syndrome, coupled with its striking phenomenology, has given rise to a variety of explanations. Psychoanalytic formulations, first mooted by Capgras & Carette (1924), are still favoured by many (Berson, 1983). Recently, however, several authors have noted that indicators of organic brain disease are often found in Capgras' syndrome patients. Electrophysiological and neuroimaging studies have shown focal or diffuse abnormalities to exist in the majority of cases investigated (Christodoulou, 1977; Lewis, 1989). The broad hypothesis arising from such reports is that the form and, to some extent, the content of the syndrome is mediated not so much by psychodynamic mechanisms as by the presence of a subtle neuropsychological impairment of visual perception and memory.

This report describes a case in which the Capgras' syndrome in a patient with clinical AIDS was associated with a right-sided neuroradiological abnormality and corresponding neuropsychological deficits.

Case report

The subject, a 30-year-old homosexual man who worked as a computer consultant, had had no previous psychiatric history and no history of drug or alcohol abuse. His elder sister had had two psychiatric admissions at the age of 37 years, when, according to the patient, she "started to take religion too seriously"; no details of her diagnosis and treatment were available.

The patient's birth followed a pregnancy during which his mother contracted rubella in the first trimester. There were no sequelae apart from some impairment of hearing in his right ear. At the age of 25 years he was ill for a month with hepatitis (type unknown) and at 26 he was treated for anal gonorrhoea. The patient remained well until aged 30 years when he developed flu-like symptoms with night sweats and a sore throat, was found to have candidiasis of the mouth and throat and treated with ketoconazole. HIV serology at that time was found to be positive. During the next three months, the candidiasis and flu-like symptoms returned and he developed respiratory symptoms for the first time with shortness of breath and coughing. He was treated with azidothymidine (AZT), but worsening symptoms led to his admission to hospital.

On admission, a clinical and radiological diagnosis of AIDS with pneumocystis pneumonia was made and the patient was treated with high doses of co-trimoxazole. He was noted to be anxious and withdrawn during the first three days of admission.

The following day his oxygen partial pressure (P-O_2) had fallen to 43 mmHg (normal range 88–115), his temperature had risen to 39.3 °C and his mental state was described as "awake, but confused": he said that everybody he knew had caught AIDS from him and would die. Cerebrospinal fluid (CSF) examination was normal. He was given 40 mg prednisolone (o.d.) over the next two days. On day six he developed floridly psychotic symptoms. Psychiatric referral was made.

Mental state examination at this time showed an anxious, perplexed, distractible patient. He would sometimes interrupt the interview by getting up to examine some detail of his room. His speech was largely normal in form, although he frequently made irrelevant comments. He could be tearful one moment and elated the next.

He admitted to several abnormal beliefs concerning the identity of people close to him and his orientation to place. When two of his colleagues visited him he was convinced that they had been replaced by alien impostors who were physically identical and were working with the IRA. These impostors had abducted him to a place which superficially resembled a hospital ward, but in reality was the mechanics' room in a garage where he was to be held and used "as a third party to communicate between earth and their own world". He explained that these impostors used a secret staircase in the wall behind his bed. He was suspicious of his mother when she visited, remarking "you look just like my mother".

He could not detect any differences in the appearance of these people, but 'knew' that they were impostors. This belief did not extend to other friends or other family members, nor to inanimate objects. In addition, he believed that the world was running out of water and oxygen, and was about to collide with the sun. He was preoccupied with "the problem no" which he explained as follows: "In the States they reverse the words so that whenever anybody talks, 'problem no' comes out instead of 'no problem'".

He experienced auditory and visual hallucinations: he could hear 'high voices' of work colleagues in the room speaking to him and to each other. He saw black spots in the air about to descend and set fire to his bedclothes.

Cognitive testing was difficult because of the patient's distractibility. He knew the year, but not the day or month. He gave the name of the hospital correctly, but added that he was in fact in a garage. His digit span was below average. He could repeat a name and address and three objects correctly, but had no recollection of them three minutes later. His insight was limited to his concern of lack of oxygen which he regarded as a global phenomenon. Neurological examination was normal and a urine drug-screen negative. He was right handed. The psychotic phenomena, including the delusional misidentification, continued for seven days before gradual improvement on 60 mg haloperidol daily in decreasing doses.

An electroencephalogram (EEG) recorded ten days after the onset of his psychosis showed bilateral posterior theta waves which were seen less frequently on a second EEG done 12 days later. There were no focal abnormalities.

A computerised tomography (CT) brain scan done two weeks after he became psychotic showed slightly widened sulci over the vertex and a small area of diminished density in the right upper medial parietal region, without mass effect or enhancement, thought to be compatible with a resolving toxoplasma, pneumocystic or cytomegaloviral (CMV) lesion. A repeat scan nine days later showed the sulcal widening to be unchanged, but there was no longer evidence of the parietal lesion, either before or after enhancement.

Neuropsychological testing was performed three weeks after the psychotic symptoms had resolved. At testing, he was alert, co-operative and fully orientated. However, his verbal and performance IQ (Weschler Adult Intelligence Scale, WAIS-R) showed a significant decline from 113 and 110 respectively pre-morbidly to 109 and 94. This was not explained by impaired motor speed, which was above average on specific testing.

Verbal memory was intact and immediate recall of simple designs was normal. However, there was a specific visuospatial deficit for recall of patterns not available for verbal coding; in particular, recognition memory for faces was borderline. Perceptual and naming skills, and frontal lobe tests were normal.

Discussion

To our knowledge, this is the first reported case of Capgras' syndrome arising in clinical AIDS. Curiously, one of Capgras' original cases was also suffering from a sexually-transmitted disorder involving the central nervous system: neurosyphilis (Capgras *et al*, 1924). It is becoming clear that, as with syphilis, the neuropsychiatric complications of HIV infection are wide-ranging. There seems to be an increased risk of paranoid psychosis, although this may reflect the high prevalence of intracranial sequelae, particularly opportunistic infection, rather than being specific to the neurotropic virus itself (Halstead *et al*, 1988).

In the case described here, the form of the psychosis was that of an acute confusional state (ICD-9 293.0; World Health Organization, 1978), with mild global impairment of cognition and consciousness, labile mood and distractibility. Pyrexia, hypoxia and evidence for an intracranial infection were all present during the acute phase, resolving as the psychosis cleared. The content of the psychosis was more unusual: there was a relatively sustained delusional misidentification syndrome with features typical of a Capgras' delusion (that familiar people had been replaced by malevolent doubles) as well as a typical reduplicative paramnesia (the belief that his surroundings had also been duplicated).

Although an object relations theorist might infer much from the vignette of a homosexual man faced with a terminal illness, all the objective evidence in this case points to a primary organic aetiology. Several features are shared with previous examples in the literature. Clouding of consciousness has been noted in several cases (e.g. MacCallum, 1973). Bilateral temporal slow waves on the EEG seem to be characteristic (Christodoulou, 1977); in this case, bilateral posterior slow wave activity was noted.

The presence of reduplicative paramnesia alongside Capgras' syndrome illustrates how closely related these two traditionally distinct disorders probably are. Reduplicative paramnesia (Pick, 1903) has been considered a neurological disorder, and Capgras' syndrome a psychiatric one. Yet, the only clinical distinction between the two disorders is whether the abnormal belief involves places or people; in fact, two of Pick's original cases misidentified both. Similar explanations have been offered for the two disorders (Staton *et al*, 1982; Lewis, 1987). In some ways, the separation of the two disorders serves only to point out the often artificial boundary between psychiatry and neurology.

The radiological and neuropsychological findings in this case suggest again that there may be a localisable disturbance of function in Capgras' syndrome. A lesion was shown in the right parietal region on CT. Right-sided pathology has previously been described on neuroimaging in Capgras' syndrome (e.g. Alexander *et al*, 1979; Burns, 1985), in reduplicative paramnesia (e.g. Kapur *et al*, 1988), and in one patient showing both (Staton *et al*, 1982). The significance of the radiological findings is supported by the demonstration on careful testing of a specific neuropsychological impairment for the recall of complex visual patterns, a function of the non-dominant parietal area. This has also been reported in previous cases of Capgras' syndrome (Christodoulou, 1977; Alexander *et al*, 1979; Lewis, 1987).

In conclusion, it seems we can add Capgras' syndrome to the lengthening list of psychiatric disturbances arising in association with AIDS. In this case, the link was probably mediated by transient right-sided parietal lesion causing a subtle visuospatial amnestic deficit.

Acknowledgement

We thank Dr W. A Seed, Reader in Medicine, for permission to report this case.

References

ALEXANDER, M. P., STUSS, D. T. & BENSON, D. R. (1979) Capgras syndrome: a reduplicative phenomenon. *Neurology*, **29**, 334–339

BERSON, R. J (1983) Capgras' syndrome. *American Journal of Psychiatry*, **140**, 969–978.

BURNS, A (1985) The oldest patient with Capgras syndrome? *British Journal of Psychiatry*, **147**, 719–720.

CAPGRAS, J. & REBOUL-LACHAUX, J. (1923) Illusion des sosies dans un delire systemise chronique. *Bulletin de la Societe Clinique de Medicine Mentale*, **2**, 6–16.

—— & CARETTE, P. (1924) Illusion des sosies et complexe d'Oedipé. *Annalès Medico-Psychologiques*, **82**, 48.

—— & LUCCINI, P. & SCHIFF, P. (1924) Du sentiment d'estrangete a l'Illusion des sosies *Bulletin de la Societe Clinique de Medicine Mentale*, **121**, 210–217.

CHRISTODOULOU, G. N. (1977) The syndrome of Capgras. *British Journal of Psychiatry*, **130**, 556–564.

DAMASIO, A. R. (1985) Prosopagnosia. *Trends in Neurosciences*, **8**, 132–133.

HALSTEAD, S., RICCIO, M., HARLOW, P., *et al* (1988) Psychosis associated with HIV infection. *British Journal of Psychiatry*, **153**, 618–623.

KAPUR, N., TURNER, A & KING, C (1988) Reduplicative paramnesia possible anatomical and neuropsychological mechanisms. *Journal of Neurology, Neurosurgery and Psychiatry*, **51**, 579–581.

LEWIS, S. W (1987) Brain imaging in a case of Capgras' syndrome. *British Journal of Psychiatry*, **150**, 117–121.

—— (1989) Capgras' syndrome. *British Journal of Psychiatry*, **154**, 270–271.

MACCALLUM, W. A. G. (1973) Capgras' symptoms with an organic basis. *British Journal of Psychiatry*, **123**, 639–642.

PICK, A. (1903) Clinical studies: III. On reduplicative paramnesia. *Brain*, **26**, 260–267.

STATON, R. D., BRUMBACK, R. A , WILSON, H. (1982) Reduplicative paramnesia: a disconnection syndrome of memory. *Cortex*, **18**, 23–26.

WORLD HEALTH ORGANIZATION (1978) *Mental Disorders: Glossary and Guide to their Classification in Accordance with the Ninth Revision of the International Classification of Diseases* (ICD-9). Geneva: WHO.

*Paul Crichton, MA, MB, MRCPsych, *Registrar, Department of Psychiatry, Charing Cross Hospital, London W6 8RP*; Shôn Lewis, BSc, MPhil, MRCPsych, *Senior Lecturer, Department of Psychiatry, Charing Cross and Westminster Medical School, London W6 8RP*

*Correspondence

British Journal of Psychiatry (1990), 157, 611–613

Rapid-Cycling Disorder in Association with Cerebral Sarcoidosis

D. G. WALBRIDGE

A 48-hour rapid-cycling disorder in a patient with pre-existing cerebral sarcoidosis, not described previously, was similar to that of others in the literature in that the mood switching occurred during sleep. Although the condition is rare, its existence raises important questions regarding the neurophysiology of affective disorders in general.

A 48-hour rapid-cycling disorder is a rare condition comprising low mood alternating with normal or elevated mood, the whole pattern repeating itself every 48 hours. Bunney & Hartmann (1965) reported a single case and reviewed the European literature from 1804 onwards, finding only ten other cases. A computerised literature search up to the present day revealed a further nine cases, making a total of 20.

Perhaps the most striking feature of the condition is the almost perfect regularity of the pattern of alternating days. Typically, the patient is described as experiencing two consecutive days of the same mood only a few times during several years of follow-up. In this report, a further case of 48-hour rapid-cycling disorder is presented in a patient with pre-existing cerebral sarcoidosis, the association having not been previously described.

Case report

Mrs Y, a 50-year-old married housewife, was referred for psychiatric assessment with a one-year history of 48-hour mood swings of gradual onset. Low days were characterised by feelings of misery, fatigue, guilt and apathy with no interest in doing more than the minimum necessary household tasks. Prominent hypochondriacal worries, thoughts of worthlessness and self-blame also present. High days found her generally over-cheerful, but prone to outbursts of tearfulness and irritability; her husband described her, in addition, as argumentative, abusive, distractible and reckless with money. There had never been psychotic phenomena, physical aggressiveness or thoughts of suicide. As far as she and her husband could remember, the pattern of alternating low and high days had remained absolutely regular over the past year.

Following a low day, she would sleep from 10 p.m. to 3 a.m. and wake feeling miserable, but looking forward to the coming high day. At some time between 7 and 8 a.m. her mood would rise to its full high-day level over a period of a few seconds. Following a high day, she would sleep from 10 p.m. to 7 a.m. and wake feeling miserable, the mood change having occurred during sleep. Her appetite was unaffected.

At the age of 30 years, she had presented with headache, oscillopsia and weakness and clumsiness of the left leg. A diagnosis of cerebral sarcoidosis was made, based on raised protein and lymphocytes in the cerebrospinal fluid, hilar lymphadenopathy on chest X-ray and a positive Kveim test. She was treated with a course of prednisolone, and her symptoms quickly resolved, leaving her with mild weakness and clumsiness of the left leg which has persisted unchanged to the present day.

At the age of 35 years she was admitted to a psychiatric hospital with symptoms of pre-senile dementia. On the assumption that the dementia was due to a recurrence of her cerebral sarcoidosis, she was treated with a short course of dexamethasone and made a rapid and full recovery.

Between the recovery from pre-senile dementia and the onset of 48-hour mood swings she had been mentally and physically well. Her husband reported that she had been cheerful and sociable in her personality with no undue tendency to anxiety or mood fluctuations. There was no personal or family history of functional psychiatric disorder and she rarely drank alcohol. Her only medication was lorazepam (0.25 mg *nocte*), which she was immediately able to stop.

Physical examination revealed upper motor neurone weakness, extensor plantar reflex and mild cerebellar ataxia of the left leg. The right eye was virtually blind, with signs of optic atrophy. Routine blood tests, including calcium series, were normal. Although thyroxin level was at the low borderline of the normal range, thyroid-stimulating hormone level was normal and there were no physical symptoms of hypothyroidism.

It was decided that steroid medication was not indicated and that the mood-swings should be treated symptomatically with either lithium carbonate or carbamazepine. Initially, the patient refused to accept medication because of the need for continued blood tests and her marked fear of hypodermic needles. With her husband's persuasion she eventually, and reluctantly, chose to try carbamazepine on the understanding that fewer tests would be needed than with lithium. At a dose of 200 mg b.i.d., her serum carbamazepine level was well within the range considered therapeutic for anticonvulsant usage. This level was maintained for a period of six months without benefit to her mood-swings and carbamazepine was therefore withdrawn.

Because of the borderline–low thyroxin level and the association between hypothyroidism and rapid-cycling mood disorder, the next strategy was to try medication with thyroxin (0.1 mg daily). This suppressed the thyroid stimulating hormone to 0.1 mU/l (normal range 0.5–6.0), but had no beneficial effect on the mood-swings either alone or during a period when thyroxin and carbamazepine were taken concurrently.

During an 18-month follow-up, the patient's physical state has not altered, the weakness of the left leg and the loss of sight in the right eye remaining the same. Unfortunately, she remains unwilling to accept medication with lithium carbonate, despite the possibility that it might have a marked effect on her mood-swings. The 48-hour cycle has been perfect in that there have never been two consecutive high days nor two consecutive low days, and a prediction made 18 months ago as to Mrs Y's mood on a given day at the present time would have been correct.

Discussion

Many of the 10 most recently described patients have been investigated in a variety of ways so that, despite the small number of cases, there is quite a large body of data (Bunney & Hartmann, 1965; Jenner *et al*, 1967; Kupfer & Heninger, 1972; Doerr *et al*, 1979; Gelenberg *et al*, 1979; King *et al*, 1979; Frank & Harrer, 1980; Paschalis *et al*, 1980; Welsh *et al*, 1986; Zisook, 1988). Six patients were men and four were women. In seven cases, the mood swings were bipolar, the remaining three alternating between low and normal mood. Three cases, all bipolar, at times exhibited symptoms of psychosis.

The time of the switch between different moods is mentioned for most cases and nearly always occurred during sleep. The speed of mood change is less well described, but at least one patient resembled the present case in that the switch occurred almost instanteously if he was kept awake. Patients with ordinary or rapid-cycling (longer than 48-hour) bipolar affective disorder are also more likely to switch between moods while asleep than while awake, especially when changing from depression to mania, and both gradual and sudden switches have been described (Wehr *et al*, 1982). Other investigations have included electroencephalograms (EEG), observation in time isolation, and the relationship between mood and corticosteroid metabolism; results, however, have not been conclusive.

In six patients, medication with lithium carbonate is mentioned and all benefited by a reduction in severity or complete abolition of their mood-swings.

Sarcoidosis affects the central nervous system (CNS) in up to 5% of cases, the cranial nerves, meninges, hypothalamus and pituitary being the common sites for lesions to occur. A variety of pictures of dementia, delirium, personality change and psychosis have been described in association with sarcoidosis, either caused directly by CNS involvement or indirectly via the effects of metabolic abnormalities or steroid medication (Lishman, 1987). However, there has been no previous description of an associated 48-hour rapid cycling.

A condition in which mood changes occur regularly and very rapidly, raises questions concerning the mechanism by which switching between depression and mania occurs. Certain physiological processes, such as the opening or closing of ionic channels or the release of stored neurotransmitter, could well take place over very short periods of time, whereas others such as the synthesis or axonal transport of neurotransmitters and receptor proteins probably could not. Results from a recent study by Carney *et al* (1989) suggest that, whatever the mechanism by which mood is maintained, the switch itself involves dopamine pathways and metabolism.

In considering the aetiology of 48-hour rapid-cycling, the central question must be whether it is simply an uncommon and extreme variant of affective disorder, or whether it is a discrete condition with superficially similar mood-related symptoms resulting from a different underlying lesion. Whatever the answer, suggestions that the biological clock systems might be involved and the predilection of sarcoid granulomas for the hypothalamus and pituitary raise the possibility that the hypothalamic-pituitary axis might be the site of the causative lesion.

Acknowledgement

I would like to thank Professor J. Newsom-Davis for reviewing the neurological history and diagnosis of this case.

References

Bunney, W. E. & Hartmann, E. L. (1965) Study of a patient with 48-hour manic-depressive cycles. Part I An analysis of behavioural factors. *Archives of General Psychiatry*, **12**, 611–618.

Carney, M. W P., Chary, T. K N., Bottiglieri, T., *et al* (1989) The switch mechanism and the bipolar/unipolar dichotomy. *British Journal of Psychiatry*, **154**, 48–51.

Doerr, P., Von Zerssen, D., Fischler, M., *et al* (1979) Relationship between mood changes and adrenal cortical activity in a patient with 48-hour unipolar depressive cycles. *Journal of Affective Disorders*, **1**, 93–104.

Frank, C. & Harrer, G. (1980) Zyklothymie mit persistierender 48-stundenperiodik. *Wiener Medizinische Wochenshrift*, **130**, 45–48.

Gelenberg, A. J., Klerman, G. L., Hartmann, E. L., *et al* (1979) Recurrent unipolar depressions with a 48-hour cycle: report of a case. *British Journal of Psychiatry*, **133**, 123–129.

Jenner, F. A., Gjessing, L. R., Cox, J. R., *et al* (1967) A manic depressive psychotic with a persistent forty-eight hour cycle *British Journal of Psychiatry*, **113**, 895–910

King, D. J., Salem, S. A. & Meimary, N. S. (1979) A 48-hour periodic manic-depressive illness presenting in late life (letter) *British Journal of Psychiatry*, **135**, 190–191.

Kupfer, D. J. & Heninger, G. R. (1972) REM activity as a correlate of mood changes throughout the night.

Electroencephalographic sleep patterns in a patient with a 48-hour cyclic mood disorder. *Archives of General Psychiatry*, 27, 368–373.

LISHMAN, W. A. (1987) *Organic Psychiatry*. Oxford: Blackwell Scientific.

PASCHALIS, C., PAVLOU, A. & PAPADIOMITRIOU, A. (1980) A stepped forty-eight hour manic–depressive cycle. *British Journal of Psychiatry*, 137, 332–336.

WEHR, T. A., GOODWIN, F. K., WIRZ-JUSTICE, A., *et al* (1982) 48-hour sleep-wake cycles in manic-depressive illness: naturalistic observations and sleep deprivation experiments. *Archives of General Psychiatry*, 39, 559–565.

WELSH, D. K., NINO-MURCIA, G., GANDER, P. H., *et al* (1986) Regular 48-hour cycling of sleep duration and mood in a 35-year-old woman: use of lithium in time isolation. *Biological Psychiatry*, 21, 527–537.

ZISOOK, S. (1988) Cyclic 48-hour depression. *Journal of Nervous and Mental Disease*, 176, 53–56.

David G. Walbridge, BSc, MBChB, MRCPsych, *Senior Registrar in Psychiatry, Littlemore Hospital, Oxford*

British Journal of Psychiatry (1990), 157, 613–616

Temporary Munchausen Syndrome

P. M. HIGGINS

Over 14 years a patient was admitted at least 91 times to at least 25 general and four psychiatric hospitals and treated for a wide range of conditions that she did not have. She appears to have recovered. The medical and psychiatric care she received probably contributed to the onset of this disorder in a person already vulnerable, and to its maintenance.

Individuals who repeatedly gain admission to hospital with dramatic but untruthful complaints of physical disease are commonly described as having 'Munchausen syndrome' (Asher, 1951). Some also simulate mental illness (Cheng & Hummel, 1978; Jones & Sternberg, 1985). Other terms used in such cases include chronic factitious illness (Spiro, 1968), hospital addiction (Barker, 1962), and hospital hoboes (Clarke & Melnick, 1958).

Nadelson (1979) regards Munchausen syndrome as a subgroup of the borderline character and distinguishes two types: those who have no settled abode and for whom simulation of illness is the centre of their lives, and those who may stay in one place for months or years and who continue to work, often in nursing or medicine.

Those exhibiting Munchausen behaviour display considerable medical knowledge (Barker, 1962) and may see doctors as objects of admiration (Cramer *et al*, 1971) or as surrogate parents compensating for an emotionally deprived childhood (Wimberley, 1981; Carney & Brown, 1983). Traumatic early admission to hospital may precede the onset of the condition (Spiro, 1968). It is generally regarded as irremediable (Ford, 1983), although a cure has been claimed in one case treated in a psychiatric hospital over three years (Yassa, 1978).

A case in which the disturbance appears to have been temporary and in which hospital care probably precipitated and reinforced the abnormal behaviour is reported.

Case report

From 1966 to 1980 Sharon K was admitted to hospital on at least 91 occasions, almost all of which followed self-referral to casualty departments. At least 25 general hospitals in and around London (including all but one of London's teaching hospitals) and at least four psychiatric hospitals were involved in her 46 admissions, 45 of which were for overdose, self-injury and a number of presumed psychiatric disorders treated by all the methods then available (Table I).

The disturbance began during Sharon's 16th year (1966), when she was admitted to her local hospital on four occasions (including twice for abdominal pain and once for 'vaginitis'), and had a normal appendix removed. The following year, she was admitted twice to the same hospital with abdominal pain and haematuria. Renal biopsy and intravenous pyelogram (IVP) were normal and she was transferred to a teaching hospital for further investigation. Medullary sponge kidney was diagnosed and she was told that she would have to take antibiotics for life. She was readmitted at the end of that year, underwent dilatation and curettage and lymphangiography, tore open the operation wound and, after leaving hospital in January

TABLE I

List of 'medical' and psychiatric diagnoses made as well as operations and treatments received, 1966–83

Diagnoses made	*Operations/treatments received*
Medical	*Operations*
Appendicitis, medullary sponge kidney, meningitis peripheral neuritis, cholecystitis[1], biliary colic, abortion (spontaneous), multiple sclerosis, salpingitis[1], endometritis, ectopic pregnancy, asthma[1], status asthmaticus[1], deep-vein thrombosis[1], pulmonary embolism[1], prolapsed disc, (pancreatitis)[2]	Laparoscopy, laparotomy, appendicectomy, cholecystectomy, dilatation & curettage[1], salpingo-oophorectomy, Daughtrey's operation, (ovarian cystectomy)[2]
Psychiatric	*Treatments*
Munchausen syndrome, hysterical personality, atypical depression, behaviour disorder, psychopathic personality, schizophreniform psychosis, anorexia nervosa	Electroconvulsive therapy (23 shocks), lithium, prolonged narcosis, psychotherapy, insulin shock, antidepressants, anxiolytics, antipsychotics

1. More than once
2. Claimed by patient only – not reported by hospital.

1968, mutilated her vagina. That year she took an overdose, inserted needles into her urethra and vagina, and cut her vagina and wrists. The ten admissions during 1968–69 include two to a psychiatric hospital, where she was treated first for two weeks and then for six months; the diagnosis on discharge was hysterical personality disorder. In 1970, after admissions to hospital for abdominal pain, loin pain, urinary infection and possibly meningitis, she was admitted to another psychiatric hospital with a diagnosis of atypical depression and anorexia; her treatment included antidepressants, phenothiazines, lithium, prolonged narcosis, insulin shocks and 23 electroconvulsive shocks. After four months she was much improved, but on the day of her discharge she cut her wrists.

Between 1971 and 1973 there were 21 known admissions to two psychiatric hospitals and to seven general hospitals, 10 for self-injury (wrists and vagina), five for abdominal pain, two for overdose, two for 'hysteria', one for dysuria and one for depression. Admissions peaked in 1974, with 14 to general hospitals and two to psychiatric hospitals. Seven admissions were for 'medical' problems: frequency and dysuria (twice), biliary colic, incomplete abortion, endometritis, facial numbness, and salpingitis. She also claimed two other admissions, for pancreatitis and for dilatation and curettage. While an in-patient she tried to strangle herself and to mutilate her vagina. This is the last year in which self-mutilation is known to have occurred.

Eleven admissions were reported in 1975. She was treated for cholecystitis on two occasions and for abdominal pain on another two occasions; four admissions were for psychiatric conditions and three were for treatment of overdose. She stated that she had had an ovarian cyst removed but no report of that admission was received.

At that stage all approaches had failed, including psychotherapy and numerous attempts to correct a presumed depressive illness. She was on increasing doses of tranquillisers and antipsychotic drugs, rising in mid-1975 to 1600 mg of chlorpromazine and 80 mg of diazepam daily and 80 mg of flupenthixol decanoate weekly. In September that year the psychiatrist arranged for her to go to a hostel since her parents could no longer cope with her and in hospital she had been destructive and manipulative.

She left the hostel in November but asked for readmission later that month as her mother refused to have her at home. She was discharged in December on flupenthixol decanoate (80 mg weekly), chlorpromazine (300 mg *nocte*), lorazepam (7.5 mg daily) and trimipramine (25 mg *nocte*). A place in another hostel was arranged but she stayed there only one day.

The 16 known admissions over the four years from 1976–79 included four for 'asthma' and/or 'pulmonary embolism', one for 'multiple sclerosis', one for investigation of abdominal pain and two unnecessary operations (cholecystectomy and salpingo-oophorectomy). She was discovered to be attending five different oral surgical clinics in London for treatment of recurrent dislocation of the jaw.

There were also signs of improvement. She had worked briefly as a student nurse in 1973 and as a hospital technician in 1976 but, in 1977, in order to work away from hospitals, she obtained a job in a local office and a better post in central London the following year. Admission was avoided three times and shortened twice during 1978; she was twice recognised and turned away from local casualty departments; her father declined to take her to hospital when she became breathless; and on two occasions, her parents insisted that hospital staff inform me of her admission. In 1980 when she developed back pain while at work she rejected an offer to take her to hospital and went home instead. There was slight stiffness of her lumbar spine. Later that year she developed back pain on a coach travelling to Rome and on arrival was admitted to hospital there. When flown home a week later examination revealed scolioisis and an X-ray showed narrowing of the disc space between L4 and L5. An orthopaedic surgeon admitted her for traction.

There were no admissions in 1981 or 1982 and consultations, which had reached a peak of 46 during 1978, fell to normal levels in 1982. She gained promotion at work and looked more relaxed. She was leading a more independent life and her father professed himself astounded at the change in her.

In 1983, just before she was about to move into the country with her parents, she said that her back was still troubling her. The lumbar spine was stiff and there was an area of sensory loss on the outer side of the right foot and ankle. Five days later admission to hospital was arranged since she had retention of urine and loss of sensation in both legs. The sensory signs disappeared soon after admission. Myelography revealed a slight bulge at the level of the disc between the fourth and fifth lumbar vetebra but the surgeon did not consider that an operation was indicated.

Some months later her new doctor indicated that she had developed acute back pain with retention following a lifting strain and a disc fragment embedded in the spinal cord had been removed. She was confined to a wheelchair and had an in-dwelling catheter. Early in 1987 she was walking with a stick. By the end of that year she was free of the catheter and had started a new career. Her mother told me that she was delighted with her progress and that all her troubles were now behind her.

Management

I became Sharon's GP in 1975. It was decided that she should be seen by only one other partner if I was unavailable. I had to gain her confidence; I explored her complaints systematically. I also tried to help her understand the origin and meaning of bodily sensations. When she manipulated, I made it clear that I knew what she was doing. If she attended hospital without my consent, I asked her to account for her actions.

From 1978, Sharon's parents actively co-operated with me and this was an important factor in changing her behaviour.

Aetiological factors

There was some evidence that Sharon was already vulnerable before her psychopathology. She was the last of three children, felt less competent than her siblings and was unhappy at school. Her classmates teased her but the school nurse was sympathetic and understanding; the medical room became a place of refuge for her.

There is nothing to suggest inadequate parenting. All three children worked in hospitals, her brother as a technician, her sister as a ward clerk and Sharon herself first as student nurse and later as a technician. Her sister had left before the family moved; her brother had a long history of pruritis of the scalp and pains in his hands and body and was described as nervous and fidgety.

What led to the first admission when she was 15 is not known. Her general practitioner's (GP's) notes record her as depressed at that time. It is possible that the haematuria which led to her being transferred to a teaching hospital followed either a sexual experience or injury (perhaps self-inflicted) to her genitalia – a part of her body that she "did not like".

She seems to have become much more disturbed after the diagnosis in 1967 of 'medullary sponge kidney' that resulted in the realisation that other dangers threatened; "hearing the Professor talking to the students about leukaemia was a big shock". She was preoccupied with her body, yet tried to distance herself from it, and could not control it. Nevertheless, she faked illness: she would wake up in the morning and decide to be ill, but when she got to hospital she would really feel ill.

More than once she expressed anxiety about her difficulty in distinguishing what was real from what was not. She experienced panics during which she only felt safe while in hospital. Events following the admission in 1967 could be viewed as repeated attempts to test bodily wholeness. Unhappily, each admission reinforced her fears. With few exceptions each putative condition elicited the full hospital response; repeatedly she learnt that it was possible to have something seriously wrong and not to know of it. The barrage of physical and chemical psychiatric treatments in 1970 probably further lessened her hold upon reality. Unfortunately, access to hospitals was easy for her. Within three miles of her home were three hospitals all with casualty departments and, within 14 miles, nine hospitals south of the Thames alone.

Discussion

This case demonstrates that Munchausen syndrome may be temporary, and that one person can mimic an astonishing range of serious medical disorders, sometimes with and sometimes without intent to deceive.

All the evidence indicates a substantial return to normality over the period of observation (Fig. 1). There were indications that she was distressed by what was happening. The massive doses of tranquillisers in 1975 may have given her a breathing space by rendering her physically incapable of making her way to hospital. The turning point came in 1978 when she was rebuffed by two of our local hospitals, when her parents altered their response to her behaviour and when she began to look to us rather than to hospitals for help (Fig. 1). Sharon attributed her recovery to faith healing at her local church and her father agreed that it had a religious origin but the psychiatrist who had befriended her had also contributed.

There were some favourable factors throughout. Although at one point disease simulation became the centre of her life, she did not wander from home, and from 1977 she was in regular employment. Apart from two short periods her parents were supportive. This stability of residence offered the opportunity for consistent management. The approach adopted was not the non-threatening relationship suggested by Ford (1983) but rather that advised by Groves (1981) for borderline personality disorders: firm, consistent, even authoritarian.

Apart from the prolapsed disc, there is no firm evidence that Sharon actually suffered from any of the medical and psychiatric conditions for which she was treated. Management of her disc problem was complicated by apparent sensory loss that was not compatible with the site of the presumed lesion and by accounts of fever that could not be confirmed.

Although individuals with Briquet's syndrome (Guze, 1975) or the 'thick file' patients so familiar to GPs also use hospital services in an excessive

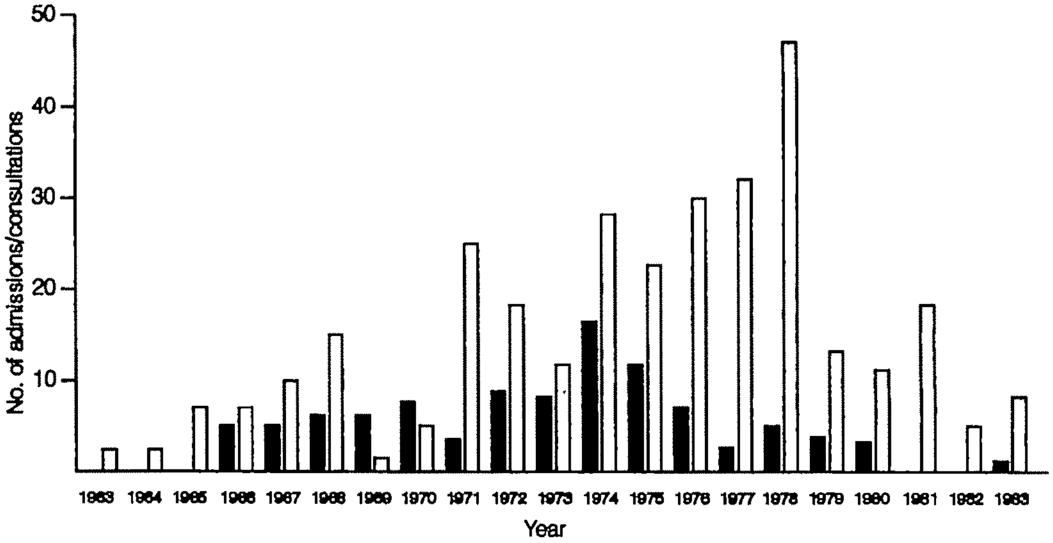


Fig. 1 Summary of case history, 1963–83, showing number of hospital admissions (■) and consultations with general practitioners (□).

and misplaced way, the apparent absence of deliberate deception differentiates such cases from Munchausen behaviour. However, this distinction is not well founded, but rests, in the case of Briquet's syndrome, on the unverifiable notion of hysteria being a wholly subconscious mechanism, and, in the case of 'thick-file' patients, on the undramatic nature of their behaviour. Simulated illness, it has been claimed, is much more common than is generally believed (Carney & Brown, 1983; Naish, 1979). Much of it is not detected and, even when doctors suspect simulation, they tend to 'play safe' and to collude with the patient (Carney & Brown, 1983). The events described here give some indication of the resultant waste of resources and of human life.

Acknowledgements

My thanks are due to Eileen Turner who searched the voluminous records and tabulated the essential facts of this case and to my two secretaries who typed endless drafts, Dorett Woodfine and Carol Butterfield.

References

Asher, R. (1951) *Munchausen Syndrome*. Lancet, *i*, 339–341.

Barker, J. C. (1962) The syndrome of hospital addiction (Munchausen syndrome). *Journal of Mental Science*, **108**, 167–182.

Carney, M. W. P. & Brown, J. P. (1983) Clinical factors and motives among 42 artifactual illness patients. *British Journal of Medical Psychology*, **56**, 57–66.

Cheng, L. & Hummell, L. (1978) The Munchausen syndrome as a psychiatric condition. *British Journal of Psychiatry*, **133**, 20–21.

Clarke, E. & Melnick, S L. (1958) The Munchausen syndrome or the problem of hospital hobos. *American Journal of Medicine*, **25**, 6–12.

Cramer, B., Gershberg, M. R. & Stern, M. (1971) Munchausen syndrome: its relationship to malingering, hysteria and the physician–patient relationship. *Archives of General Psychiatry*, **24**, 575–578.

Ford, C. V. (1983) Munchausen syndrome. In *The Somatising Disorders: Illness as a Way of Life*. Oxford: Elsevier Biomedical.

Groves, J. E. (1981) Borderline personality disorders. *New England Journal of Medicine*, **305**, 259–262.

Guze, S. B. (1975) The validity and significance of the clinical diagnosis of hysteria (Briquet's syndrome). *American Journal of Psychiatry*, **132**, 138–141.

Jones, E. M. & Sternberg, M. P. (1985) Muchausen syndrome (letter). *British Journal of Psychiatry*, **147**, 729–730.

Nadelson, T. (1979) The Munchausen spectrum: borderline character features. *General Hospital Psychiatry*, **1**, 11–17.

Naish, J. M. (1979) Problems of deception in medical practice. *Lancet*, *ii*, 139–142.

Spiro, H. R. (1968) Chronic factitious illness (Munchausen syndrome). *Archives General Psychiatry*, **18**, 569–579.

Wimberley, T. (1981) The making of a Munchausen. *British Journal of Medical Psychology*, **54**, 121–129.

Yassa, R. (1978) Munchausen's syndrome: a successfully treated case. *Psychosomatics*, **19**, 242–243.

P. M. Higgins, OBE, MB, BS, FRCP, FRCGP, *Emeritus Professor, University of London; Wallings, Heathfield Lane, Chislehurst, Kent BR7 6AH*

British Journal of Psychiatry (1990), **157**, 617–626

Correspondence

Editor: Ian Pullen

Contents: Neuroleptic malignant syndrome/Calcium-channel blockade and depressive illness/Investigating psychosurgery/WHO consensus statement/Benefits of routine laboratory investigations/Dyskinesia in the mentally handicapped/Linguistics of schizophrenia/Understanding Capgras syndrome/Multifactorial intoxication?/Memories in depression: pleasant or unpleasant?/Expressed emotion and lithium prophylaxis/Pseudocyesis followed by depressive psychosis/Ethics of 'brain transplants'.

Neuroleptic malignant syndrome

Sir: The wide spectrum of syndromes associated with neuroleptic chemotherapy has elicited numerous case reports documenting the neuroleptic malignant syndrome (NMS). Epidemiological arguments support the notion that case reports are subject to bias and often may render misleading or partial evidence, particularly in that we are currently lacking reliable measures to differentiate NMS from the spectrum of neuroleptic-induced extrapyramidal syndromes. In most cases of suspected NMS, the diagnosis will not be ultimately confirmed. The differential diagnosis of NMS, furthermore, presents the clinician with challenging issues. Our own clinical practice suggests that severe extrapyramidal syndromes (EPS) without fever are more common than neuroleptic-associated febrile syndromes. Patients with EPS and fever often demonstrate concurrent medical disorders. In the absence of known medical aetiology, we suggest erring on the side of caution by discontinuing neuroleptics temporarily while treating the EPS and medically evaluating the patient. The usefulness of laboratory tests, in general, lies in their role as part of extensive investigations to rule out other aetiologies of fever, altered mental status, autonomic dysfunction and extrapyramidal signs. Elevated levels of creatine phosphokinase may involve several other contributing factors such as parenteral injections, other hyperthermic syndromes, agitation, and exacerbations of psychotic episodes. Our major emphasis has been on the rapid and vigorous treatment of severe extrapyramidal rigidity with or without fever. The patient who remains agitated, and becomes diaphoretic and rigid, may develop a temperature and elevated enzyme levels as a result of untreated rigidity.

While NMS is probably more directly related to central dopamine blockade and subsequent muscle contraction, we do not recommend dantrolene sodium as a first-line treatment for suspected NMS or severe EPS. We recommend dantrolene only as an alternative when anticholinergic agents or dopamine agonists are not effective. Peet & Collier (1990) present a case with apparent neuroleptic-induced rigidity associated with hyperthermia, change in mental status and autonomic dysfunction. There was no effort made to treat this patient vigorously with anticholinergic agents or dopamine agonists. Instead, a diagnosis of NMS was suspected without an apparent extensive medical investigation; the patient was treated with dantrolene and still remained rigid for one week. Most phenomena associated with NMS can be explained on the basis of the pharmacology of the drugs. Neuroleptics inhibit the dissipation of heat and may potentially cause disturbed hypothalamic temperature regulation. Some patients are apt to be treated with high dosages of neuroleptics, remain agitated, become diaphoretic and rigid, and develop a temperature – ergo, that is the NMS. Since we object to the term 'NMS' being used for a constellation of signs and symptoms that have not been specifically shown to be 'malignant', we urge all clinicians to understand what is really going on – namely, in most instances, a decompensated physiological state of the patient, pseudo-Parkinsonism, and agitation that is inadequately treated.

Velamoor *et al* (1990) report on three cases of 'incipient' NMS. There would seem to be no advantage in considering these cases as incipient forms of NMS, as this adds to the already ill-defined nature of this syndrome. Cases one and two were clearly manic. Some authors have suggested that patients with affective illness, particularly acute manic episodes with heightened metabolism, may be more susceptible to toxicity from neuroleptics and/or lithium (Addonizio *et al*, 1986; West & Meltzer, 1979). Case one was also dehydrated with hypernatraemia – a treatable concominant. Leukocytosis is a non-specific finding that may reflect recent lithium usage, stress and/or infection. This patient remained afebrile throughout and was appropriately treated with rehydration. In addition, the patient was not reported to have rigidity in the case study, despite indications of rigidity in the discussion. It is often difficult to evaluate whether certain physiological states are secondary to NMS or specific triggering factors.

Case two had confusion with increased levels of creatine phosphokinase. His pulse, blood pressure and temperature were monitored, but no baseline values were given, and no neurological abnormality reported at the time of symptomatic treatment. Case two, the only patient with documented rigidity and fever, was treated with diazepam. This patient was a 61-year-old male who received a total of 15 mg of haloperidol (750 mg of chlorpromazine equivalents). In addition, he had cardiac disease, apparent pre-renal azotemia secondary to dehydration, electrolyte imbalance and was eventually successfully rehydrated. The physiological concomitants of ageing were not kept in mind and one may question the dosage of neuroleptic in this frail individual. We can speculate that his fever was a coincidental occurrence in a dehydrated patient. He simply had extrapyramidal side-effects that were precipitated by neuroleptics.

The main emphasis is that specific criteria and a precise delineation for the diagnosis of NMS remains controversial with some question as to the existence of a specific disorder. It would be unfortunate if preoccupation with the beguiling designation 'incipient NMS' or even the full blown phenomenon of NMS sidetracks or distorts our perception of what is really going on – that is, in most cases, the concomitants of physical illness and, most importantly neuroleptic-induced rigidity possibly associated with akathisia that is inadequately treated.

Silvia Gratz
George Simpson
The Medical College of Pennsylvania
Department of Psychiatry
3200 Henry Avenue
Philadelphia
Pennsylvania 19129

References

Addonizio, G., Susman, V L. & Roth, S. D. (1986) Symptoms of neuroleptic malignant syndrome in 82 consecutive inpatients. *American Journal of Psychiatry*, **143**, 1587–1590

Peet, M. & Collier, J (1990) Use of carbamazepine in psychosis after neuroleptic malignant syndrome *British Journal of Psychiatry*, **156**, 579–581.

Velamoor, V. R , Fernando, M. L. D. & Williamson, P (1990) Incipient neuroleptic malignant syndrome. *British Journal of Psychiatry*, **156**, 581–584

West, A. P & Meltzer, H. Y (1979) Paradoxical lithium neurotoxicity. a report of five cases and a hypothesis about risk for neurotoxicity. *American Journal of Psychiatry*, **136**, 963–966

Calcium-channel blockade and depressive illness

Sir: It was with great interest that I read Eccleston & Cole's report of treatment-resistant depression associated with the use of a calcium-channel blocker (*Journal*, June 1990, **156**, 889). The authors suggest that it may be possible to explain this action of dihydropyridines in terms of the monoamine hypothesis on the basis of alterations in intraneuronal concentrations of calcium. This could be mediated by dihydropyridines reducing calcium influx through voltage-sensitive calcium channels, and noradrenaline and 5-hydroxytryptamine (5-HT) altering levels of inositol 1,4,5-triphosphate (IP3) and diacylglycerol, which regulate release of sequestered calcium from intracellular organelles. However, the spatial distribution of calcium in the cell may be more important than the average cell concentration for the control of many cellular functions. For example, it has been demonstrated that transmitter release may require calcium entry through voltage-sensitive calcium channels increasing the levels of calcium just inside the membrane, rather than general cellular increases of calcium mediated by IP3 (O'Sullivan *et al*, 1989). The fact that dihydropyridines and monoamines can both affect intraneuronal calcium may not alone be explanation enough. Behavioural studies have clearly shown that calcium-channel blockers are able to affect 5-HT_{1A} and 5-HT_2 function in rats, effects which are reported to be independent of whole-brain 5-HT synthesis and potassium-induced release from *in-vitro* brain slices (Green *et al*, 1990). Where exactly these effects are mediated is open to debate. Since the effects of calcium-channel blockers could not be reversed by using a calcium-channel agonist (Bay K 8644), Green *et al* suggest that the changes in 5-HT function are not mediated at the calcium-channel level. However, only low doses of Bay K 8644 were used because of its toxicity.

Recent electrophysiological work may be able to shed light on the situation. 5-HT has been shown to be able to directly reduce calcium currents in dorsal raphe cells of rats, an effect that is independent of its previously well characterised inhibition mediated via potassium channels (Penington & Kelly, 1990) The receptor involved appears to be the 5-HT_{1A}

presynaptic autoreceptor. This is interesting since this receptor has been implicated as a possible locus for the action of antidepressant treatments (Goodwin *et al*, 1985). In addition, electrophysiological evidence suggests that chronic antidepressants lead to a 'down regulation' of 5-HT$_{1A}$-mediated inhibition (Blier *et al*, 1988). While this has yet to be confirmed for the action of 5-HT on calcium currents, if it is, then clearly antidepressants have an opposing action to calcium-channel blockers by being able to decrease the ability of 5-HT to reduce calcium influx. Therefore the common pathway for the action of dihydropyridines and monoamines in affective disorders could be at the level of the calcium channel itself.

R. H. McAllister-Williams
Department of Pharmacology
University of Edinburgh Medical School
1 George Square
Edinburgh EH8 9JZ

References

Blier, P., de Montigny, C. & Chaput, Y (1988) Electrophysiological assessment of the effects of antidepressant treatments on the efficacy of 5-HT neurotransmission. *Clinical Neuropharmacology*, **11**(suppl. 2), S1–S10

Green, A. R., De Souza, R. J., Davies, E. M., *et al* (1990) The effects of Ca^{2+} antagonists and hydralazine on central 5-hydroxytryptamine biochemistry and function in rats and mice. *British Journal of Pharmacology*, **99**, 41–46.

Goodwin, G. M., De Souza, R. J. & Green, A. R. (1985) Presynaptic serotonin-mediated response in mice attenuated by antidepressant drugs and electroconvulsive shock. *Nature*, **317**, 531–533.

O'Sullivan, A. J., Cheek, T. R., Moreton, B., *et al* (1989) Localization and heterogeneity of agonist-induced changes in cytosolic calcium concentration in single bovine adrenal chromaffin cells from video imaging of fura-2. *The EMBO Journal*, **8**, 401–411.

Penington, N. J. & Kelly, J. S. (1990) Serotonin receptor activation reduces calcium current in an acutely dissociated adult central neuron *Neuron*, **4**, 751–758.

Investigating psychosurgery

Sir: I was interested to read Al-Sheikhli's letter (*Journal*, June 1990, **156**, 903) and, like him, I wondered why psychosurgery had not been considered in Paykel's review of the relevance of research for the treatment of depression. Professor Paykel helpfully replied (*Journal*, June 1990, **156**, 904) and pointed out that psychosurgery, while having a place with severe resistant depression, needs more research into efficacy and indications.

However, in this letter my main purpose is to question Professor Paykel's statement that "I understand that the Mental Health Act Commission has comprehensive records ...". The Commission have been involved with a total of about 100 patients and the Act requires only opinions in writing from three professionals for each patient. By contrast we have now carried out over 1200 operations since the mid-1960s and we have increasingly detailed information on all these patients over this period of time. Our problem is that money for research is not at all easy to obtain but, given better computer facilities, we have the richest possible information available for investigating psychosurgery.

Professor Paykel suggests that the College could take up the fact that more research into the efficacy and indications of psychosurgery is needed. At the foundation of the College a research committee was set up to investigate the value of psychosurgery. One of the main problems was that there seemed to be no treatments that could be used as a control. A protocol was suggested (Research Committee, 1977) but funding proved impossible.

P. K. Bridges
The Geoffrey Knight Unit for Affective Disorders
Brook General Hospital
London SE18 4LW

Reference

Research Committee, The Royal College of Psychiatrists (1977) Evaluation of the surgical treatment of functional mental illness: proposal for a prospective controlled trial. In *Neurological Treatment in Psychiatry, Pain and Epilepsy* (eds W H Sweet, S. Obrador & J. G. Martin-Rodriguez). Baltimore: University Park Press.

WHO consensus statement

Sir: We read with interest the WHO consensus statement on prophylactic use of anticholinergics in patients on long-term neuroleptic treatment (*Journal*, March 1990, **156**, 412). However, like Barnes (*Journal*, March 1990, **156**, 413–414), while we welcomed the statement, we also felt that it was too brief and failed to comment on some benefits of anticholinergic therapy. Such benefits may be of particular relevance in third-world countries where, for example, patients may experience extreme transport difficulties in reaching a hospital should acute neurological side-effects arise. The occurrence of an otherwise preventable acute dystonia may well exacerbate natural fear of 'western' medicine, so adversely affecting future compliance and willingness to return for further psychiatric care. Also, use of those antipsychotics which are less likely to produce side-effects may not be possible in situations where poor availability and relative costs must be taken into account. Other than from Morocco, there were no other African contributions to this WHO statement which may possibly explain why such factors were not considered.

We have since drawn up our own guidelines for the use of anticholinergics in patients on neuroleptics. These may be of interest to those working in similar settings and are as follows.

(a) Routine use is not recommended.

(b) At the *onset* of neuroleptic treatment where prescribing for *in-patients*, anticholinergics should only be used in the presence of acute dystonia and should be gradually withdrawn after one week.

(c) At the *onset* of neuroleptic treatment where prescribing for *out-patients*, anticholinergics should be given for two weeks where: (i) the patient is a younger male or a child; (ii) there is a past history of drug-induced dystonia; or (iii) the patient lives a considerable distance from psychiatric care.

(d) During longer-term neuroleptic treatment, anticholinergics should be prescribed only where Parkinsonism has developed and where reduction of neuroleptic dose has been ineffective. In these cases anticholinergics should be given gradually but withdrawn after two months. If symptoms persist it may be sufficient to prescribe anticholinergics strictly for one week after each depot injection. In a small number of patients with disabling bradykinesia, long-term anticholinergics will be needed in combination with the lowest possible dose of neuroleptic.

M. A. Abas
S. W. Acuda
J. C. Broadhead
I. V. Chagwedera
F. B. Chikara
J. M. Piachaud
M. Stefanovic
J. Vermeulen

Department of Psychiatry
University of Zimbabwe
PO Box A178
Avondale
Harare, Zimbabwe

Benefits of routine laboratory investigations

Sir: White & Barraclough (*Journal*, July 1989, **155**, 65–72) concluded that only very limited routine laboratory testing was justified for adult psychiatric admissions. We examined the results of laboratory screening tests, ordered on admissions to our in-patient inner-city general psychiatry unit, to determine the frequency of abnormal results and their clinical significance, and compared our results with those of Drs White & Barraclough. The patient population was primarily male veterans with substance use, psychotic and major mood disorders. A total of 2308 tests on 52 admissions of 50 patients were examined. Each admission had an average of 44.4 tests and 5.5 abnormalities; each patient had at least one abnormal result. A total of 288 (12.5%) of the tests ordered were abnormal (a figure not unlike the 10.2% reported by White & Barraclough). No unexpected physical condition responsible for psychiatric symptoms was detected by the tests.

Of the abnormal results, 26 were positive toxicology screens in 20 patients with an already known substance use diagnosis (and were hence not unexpected results), and four were from a delirious patient with known chronic renal failure. A substance use diagnosis (including alcohol use disorders) accounted for 80 of the abnormal tests, including not unexpected elevations in liver enzymes, macrocytosis, elevated mean corpuscular haemoglobin concentration, bilirubin and low albumin, platelets or folate. Eleven abnormalities seemed to be related to poor nutrition (e.g. ketonuria on urinalysis, decreased blood urea nitrogen and creatinine), and 30 seemed to be associated with dehydration. Eighteen abnormalities were in AIDS patients and were important in monitoring their azidothymidine treatment. One psychotic patient was slightly hyponatraemic, probably secondary to psychogenic polydipsia. The remainder of the abnormalities were classified as without clear significance or diagnostic relevance.

We agree with Drs White & Barraclough that most of the abnormalities were predictable and do not appear helpful in establishing previously unsuspected organic causes for psychiatric symptoms However, 170 (60%) of them were felt to be clinically significant (i.e., useful in treatment planning), as they: (a) had utility as a gauge of the severity of the patient's psychiatric condition (e.g. dehydration in a psychotic or depressed patient or liver damage in an alcohol abuser); (b) were useful in confronting the denial commonly seen in alcohol and substance abusers (e.g. using elevated liver enzymes in an alcoholic, positive toxicology screen results in a known drug-abuser denying recent use); (c) were useful in the detection of use of drugs other than those acknowledged by the patient; or (d) were important in managing various organic medical and psychiatric therapies. We would add these to the list of laboratory test benefits outlined by Drs White & Barroclough. On the basis of our study, we agree with Drs White & Barraclough's suggestion for only a selective laboratory screen on admission. The only aspect of laboratory screening on admission that should be 'routine' for the experienced psychiatric clinician is the informed, rational use of any test that is ordered.

PETER KAMP
RICHARD B. ROSSE
Washington Veterans Administration Medical Center
Psychiatry Service
50 Irving Street NW
Washington, DC, 20422

Dyskinesia in the mentally handicapped

SIR: It was very interesting to read the well designed study of a well defined population by Dinan & Golden (*Journal*, July 1990, **157**, 131–132).

We have undertaken the screening of the whole mentally handicapped population at Cell Barnes Hospital ($n=550$) for dyskinesia. After an extensive trial period we decided to use the Dyskinesia Identification System Condensed User Scale (DISCUS) instead of the Abnormal Involuntary Movement Scale (AIMS) because of the former's superiority in identifying facial tics and grimaces, and in having separate items for the tongue movements, tongue thrust, tongue tremor and athetoid/myokymic/lateral tongue (Sprague *et al*, 1989). Also, DISCUS is a standardised and validated scale for mentally handicapped people who offer little or no co-operation.

In our pilot study, the prevalence of orofacial dyskinesia in the institutionalised mentally handicapped population was 77%, in accordance with Dinan & Golden's results. Richardson *et al* (1986) found that their group of mentally handicapped people with tardive dyskinesia was characterised by a 92% rate of buccolingual masticatory movements.

We believe that large multicentre trials on mentally handicapped people with various psychiatric and drug histories, comparisons of various dyskinesia scores, and observations of affective changes in relation to dyskinesia should be the next step forward in the attempt to elucidate the pathophysiology of dyskinesia.

D. KOHEN
Charing Cross/Westminster Medical School
Unit of Mental Handicap
London W6

G. MATHEW
Cell Barnes Hospital
Highfield Lane
St Albans, Hertfordshire AL4 0RG

References

RICHARDSON, M. A, HAUGLAND, G, PASS, R, *et al* (1986) The prevalence of Tardive Dyskinesia in mentally retarded population *Psychopharmacology Bulletin*, **22**, 243–249.

SPRAGUE, R L., KALACHNIK, J. E. & SHAW, K M. (1989) Psychometric properties of the Dyskinesia Identification System: Condensed User Scale (DISCUS) *Mental Retardation*, **27**, 141–148

Linguistics of schizophrenia

SIR: Thomas *et al* (*Journal*, February 1990, **156**, 204–210) used methods of linguistic analysis to examine the free speech of schizophrenics and others, finding that three of the 16 descriptive variables which discriminated between clinical groups were directly concerned with 'embedding'. In an experimental study designed to investigate the psychological processes involved in the comprehension of sentences, Huff and I systematically varied 'degree of embedding' to generate 12 sentence-types with varying degrees of difficulty (Thomas & Huff, 1971).

Each sentence specified the rooms occupied by four people (Doctor, Grocer, Lawyer, Teacher) in a house containing six numbered rooms, two on each floor. While a sentence was displayed, the subject had to put dummy 'people' into an up-ended box with six compartments so as to depict its meaning. The time taken by the subject to "do his best" (T) and the number of 'people' correctly placed (C) were averaged for each sentence-type, for two groups of subjects separately and combined. The subjects were eight young adults with acute schizophrenic or schizophrenia-like illnesses and eight normal young adult controls.

High negative correlations (-0.85 to -0.95, $P<0.001$) were consistently found between C and T. This relationship was linear and essentially the same for patients and controls. The rank-order of difficulty for the sentence-types, with either C or T as criterion, was similar for the two groups. Evidently a single aspect of sentence structure was causing most of the difficulty, in broadly similar ways for patients and normals, although the patients performed much worse than the normals.

As the effect of 'embedding' is to make a sentence more parenthetical, we next assumed that different sentence structures imposed different amounts of 'parenthetical load' (PL) on the subject by making greater or smaller demands on the decoding processes required to extract their meaning; and secondly, that patients were more vulnerable to PL than normals. Various assumptions could be made as to the nature of the decoding processes, and each combination of assumptions (each 'model') dictated its own procedure for calculating the PL of any given sentence. It was consistently found that C correlated negatively and T positively with PL; but one model was significantly 'better' than the rest, i.e. it gave higher correlations, presumably because it best represented the characteristics of the decoding system.

We concluded that the comprehension of a self-embedded sentence such as *"The teacher, above whom the grocer, beside whom the lawyer, above whom the doctor lives, lives, lives, lives in room six"* requires a left-to-right, start-to-finish scan of the text; that each time a clause is interrupted by the beginning of another, parenthetically interpolated clause, a new temporary store is opened in memory for the new clause while the previous store remains open but quiescent; and that while material is being placed in an active store this generates interference in any previously opened, quiescent stores. In this way 'noise', arising as a consequence of the sentence structure and the way it is handled, causes the subject to make errors and also to allocate more time to the task. Individuals with intrinsically disorganised, 'noisy' minds would be especially vulnerable to this effect.

Similar mechanisms would presumably be engaged in the planning of sentences about to be uttered; so that people with 'cooler', less noisy minds should be capable of generating more complex, more parenthetical sentences; although they might not be motivated to do so and in terms of style and comprehensibility it might well be undesirable.

From this standpoint, the results of Dr Thomas *et al* would imply that chronic schizophrenics, although often less behaviourally disturbed than acute schizophrenics, have the more disordered, 'noisy' minds; that longer education is associated with less, and greater age with more, inherent susceptibility to parenthetical load and so on.

Parentheses are interruptions of a special kind. Dealing effectively with PL calls for the ability to place one or more uncompleted jobs 'on hold' in separate mental compartments and to insulate these as far as possible from mental activity elsewhere. This ability must be required for many kinds of thinking and in many everyday situations, as well as for handling language. A deficiency in this regard, if it is not the underlying cause of all thought disorder, must surely be an important component of it. A simplified version of our task, perhaps administered and scored by a small computer, could be of use in a variety of clinical, developmental and educational settings. It would be less time-consuming than an exhaustive linguistic analysis while still capturing much the same aspect of thought disorder, whether normal or psychotic in degree.

HUGH B. G. THOMAS

Middlewood Hospital
Sheffield S6 1TP

Reference

THOMAS, H. B. G. & HUFF, F. J. (1971) Comprehension and recording-time of transformed sentence. *Language & Speech*, **14**, 352–372

Understanding Capgras syndrome

SIR: I was interested to read the report by Rastogi (*Journal*, June 1990, **156**, 883–884) but disappointed to see once again the delusion of inanimate doubles explained away by psychological symbolism. This variant of the Capgras phenomenon is of particular significance for our understanding of delusional misidentification syndromes (Anderson, 1988).

I have seen three cases of Capgras in elderly patients during the last 12 months who all subsequently developed dementias. Global cognitive impairment had not been clinically apparent at presentation although mild impairment was demonstrated by detailed neuropsychological testing in case three.

Case one, a 76-year-old woman, developed delusional replacement of her husband three weeks after collapse, and her later cognitive decline following further collapse with right homonomous hemianopia suggested a multi-infarct pathology. Case two, an 84-year-old woman, insisted her entire home had been replaced by an identical home, and showed me each room in the house containing furniture identical to her own which belonged to the delusional owner of the house. Case three, a 79-year-old woman, refused to eat with her daughter who she maintained was an identical imposter. Cases two and three went on to develop gradually progressive cognitive decline suggestive of an Alzheimer process. Similar cases of Capgras have been described in association with dementias (Kumar, 1987; Lipkin, 1988) and most psychogeriatricians see such cases with some regularity.

An organic basis for the Capgras phenomenon is becoming increasingly likely (Cummings, 1985; Lewis, 1987; Anderson, 1988), and the continued preoccupation with psychodynamic formulations which have not furthered our understanding of these interesting conditions over the last 60 years is unhelpful and unrewarding.

D. N. ANDERSON

Fazakerly Hospital
Longmoor Lane
Liverpool L9 7AL

References

ANDERSON, D N. (1988) The delusion of inanimate doubles: implications for understanding the Capgras phenomenon *British Journal of Psychiatry*, **153**, 694–699.

CUMMINGS, J. L (1985) Organic delusions. phenomenology, anatomical correlations and review *British Journal of Psychiatry*, **146**, 184–197

KUMAR, V. (1987) Capgras syndrome in a patient with dementia *British Journal of Psychiatry*, **150**, 251

LEWIS, S W (1987) Brain imaging in a case of Capgras syndrome. *British Journal of Psychiatry*, **150**, 117–121.

LIPKIN, B (1988) Capgras syndrome heralding the development of dementia. *British Journal of Psychiatry*, **153**, 117–118.

Multifactorial intoxication?

SIR: I read with interest the article by Peh *et al* (*Journal*, June 1990, **156**, 891–893). However I fear that the discussion of possible mechanisms is perfunctory and potentially misleading.

Hyponatraemia is a relatively common finding in clinical practice, and numerous possible causes have been recognised (Foote, 1990). Spurious analytical errors may occur either from 'drip artefacts' or from conditions of hypertriglyceridaemia or hyperproteinaemia. Convincing evidence against these potential errors is lacking in the case described.

The limited clinical data given indicate the presence of a cardiomyopathy in a relatively young woman, apparent from early 1984, and the injudicious use of small quantities of intravenous fluid is dangerous, with disastrous consequences in this case. Biochemically, a picture of hypervolaemic hyponatraemia would be expected.

The syndrome of inappropriate antidiuretic hormone (SIADH) is rightly discussed, albeit briefly, by the authors. However no mention is made of tolbutamide's recognised ability as a sulphonylurea to induce this endocrinological abnormality. Moreover, head injury, convulsions and 'psychosis' are all described as individual causes of the same syndrome of excess antidiuretic hormone secretion in the face of low serum osmolality.

I would propose that the reasons for this unfortunate woman's hyponatraemia are at least multifactorial rather than isolated water intoxication as suggested.

ROBERT COLGATE

East Glamorgan General Hospital
Church Village
Pontypridd CF38 1AB

Reference

FOOTE, J W. (1990) Hyponatraemia diagnosis and management. *Hospital Update*, **16**, 248–258.

Memories in depression: pleasant or unpleasant?

SIR: It is generally accepted that depression is associated with unpleasant memories which are congruent with the affective state (e.g. Dunbar & Lishman, 1984; Teasdale & Dent, 1987). However, we have repeatedly been finding contradictory results regarding memory while studying other phenomena in pre-electroconvulsive therapy (ECT), drug-free, depressed (DSM–III–R diagnosed) patients admitted to hospital. For example, we unexpectedly found that over a period of 72 hours, depressives forgot significantly ($F = 6.283$, d.f. 1,20: $P < 0.021$) more negative than positive affect words, as compared with normals and schizophrenics (e.g. Calev, 1988).

In another study (Bachar *et al*, 1987), pre-ECT depressed patients tended to produce, during a reminiscing interview (encouraging subjects to speak about events which happened five years or more previously), more positive memories (mean = 51%; s.d. = 37) than did normal controls (mean = 44); s.d. = 15), although this difference was not significant. In a reminiscing group (Bachar, submitted), depressed patients produced a mean of 83% (s.d. = 18) positive affect memories. In a study of dream recall (Bachar, in preparation), we found that depressives tended to recall more positive than negative dream contents. Twenty-one out of 37 dreams were classified as positive (using Hole & Castle's (1966) classification system), and only 16 dreams were classified as negative. While dream recall norms are lacking, this finding once again shows that depressives tend to recall more positive than negative contents.

The common denominator of all our findings, in contrast to most former findings, is *delayed testing* for the 'to-be-remembered' materials. It is thus possible that positive emotional contents may be suppressed and temporarily unretrievable during depression because of state-dependent negative thought-processes. These negative materials may be well consolidated in memory, as shown by our paradigms (delayed recall, remote memory reminiscing, and dream recall). Depressives may retrieve these memories later and use them as a means for recovery.

EYTAN BACHAR
AVRAHAM CALEV
BARUCH SHAPIRA
BERNARD LERER

Sarah Herzog Memorial Hospital
The Hebrew University Hadassah Medical School
PO Box 140
Jerusalem 91001

References

BACHAR, E., DASBERG, H. & LERER, B (1987) Remembering in depressed aging patients: effect of ECT and tricyclic antidepressants – implication for psychotherapy. Paper presented at the International Conference on New Directions in Affective Disorder, Jerusalem, Israel.

CALEV, A. (1988) Effect of emotion on the rate of forgetting in normals, schizophrenics and depressives Paper presented at the First International Conference on Individual Differences, Tel Aviv, Israel.

DUNBAR, G. C & LISHMAN, W A (1984) Depression, recognition memory and hedonic tone: a signal detection analysis. *British Journal of Psychiatry*, **144**, 376–382

HOLE, C. & CASTLE, R. (1966) *Content Analysis of Dreams* New York. Appleton-Century-Crofts.

TEASDALE, J. D. & DENT, J. (1987) Cognitive vulnerability to depression· an investigation of two hypotheses *British Journal of Psychiatry*, **26**, 113–126

Expressed emotion and lithium prophylaxis

SIR: In a previous study (Priebe; *Journal*, March 1989, **154**, 396–399), expressed emotion (EE) in key relatives of 21 patients with bipolar affective or schizoaffective psychoses was assessed by the Camberwell Family Interview (CFI). All patients had been on prophylactic lithium for at least three years. Patients living with high-EE relatives showed a significantly poorer response during the three years before interview and particularly during the nine-month follow-up.

Twenty-eight months after the initial CFI, the key relatives of 15 patients were re-interviewed. All 15 patients had continued on prophylactic lithium throughout the 28 months. Two critical remarks designated high EE. There were 10 relatives identified with high EE in the first interview and eight in the second, since two relatives changed from high- to low-EE status. The course of patients' illness was assessed by means of a morbidity index (Coppen *et al*, 1973) reflecting severity and length of recurrences (a recurrence was defined by hospital admission or a temporary additional antidepressive or neuroleptic medication).

Regardless of whether EE status was defined according to first or second CFI, morbidity indices concerning the 28-month period were more than six times higher in patients living with high-EE relatives (regarding the first CFI: $t=2.91$, $P<0.05$; regarding the second CFI: $t=3.83$, $P<0.01$). Four out of five patients with consistently low-EE relatives, and one patient with a relative who had changed from high- to low-EE status were virtually without any recurrences during the 28-month period. This applied to none of the patients living with consistently high-EE relatives.

A clear answer as to whether high-EE status of relatives leads to an unfavourable course of illness or vice versa was not found. Both course of illness and relatives' EE status might be influenced by interactional patterns in the patients' families, and by changes in those patterns. As far as this small and highly selective sample is concerned, it may be concluded that patients living with consistently low-EE relatives rarely need therapeutic interventions of whatever kind in addition to prophylactic lithium.

S. PRIEBE

Department of Social Psychiatry
Freie Universität Berlin
Platanenallee 19
D 1000 Berlin 19

C WILDGRUBE

Department of Clinical Psychiatry
Freie Universität Berlin

Reference

COPPEN, H., PEET, M , BAILEY, J. *et al* (1973) Double-blind and open prospective studies of lithium prophylaxis in affective disorders *Psychiatry, Neurology and Neurosurgery*, **76**, 501–510

Pseudocyesis followed by depressive psychosis

SIR: Pseudocyesis is a false belief in pregnancy associated with its symptoms and signs (Cohen, 1982). It may be associated with psychiatric disorder, most commonly depression, which usually does not reach case level (Brown & Barglow, 1971). Occasionally, it is associated with psychosis (Taylor & Kreeger, 1987; Mortimer & Banberry, 1988; Milner & Hayes, 1990). We would like to report the first case of depressive psychosis following pseudocyesis after an interlude of normality.

Case report A 38-year-old married domestic worker of stable pre-morbid personality had children of ages 20, 14 and 10 years. She had had one miscarriage at age 35 years and one elective abortion when 37 years old. Six months after this abortion she was referred to the psychiatric services because she believed herself to be pregnant, despite two negative pregnancy tests. She had missed two periods, had back-ache, breast swelling and tenderness and morning nausea. Examination revealed a distended abdomen but non-pregnant cervix and uterus. After three weeks of supportive psychotherapy all symptoms resolved. She remained completely well for three weeks but then became depressed, with early morning wakening, reduced energy, poor appetite and weight loss. She was severely agitated, believing herself to be in danger She was deluded that her face was being distorted and that her body was rotting. She made three suicide attempts. Her symptoms failed to respond to antidepressants and phenothiazines but resolved rapidly with a short course of electroconvulsive therapy. She returned to her pre-morbid functioning and has remained well for two years off all medication.

Our patient's illness satisfied DSM–III–R criteria for a major depressive episode with psychotic features and ICD–9 guidelines for manic-depressive psychosis, depressed type. To our knowledge, this is the first report of a depressive psychosis following pseudocyesis and is the first case to demonstrate an interval of normality between resolution of pseudocyesis and onset of psychotic symptoms. This case highlights the necessity for adequate follow-up of patients with pseudocyesis: severe psychiatric illness may supervene even after a period of normality.

BRIDGET CRADDOCK
NICK CRADDOCK
L. I. LIEBLING

Highcroft Hospital
Highcroft Road
Erdington
Birmingham B23 6AX

References

BROWN, E & BARGLOW, P. (1971) Pseudocyesis: a paradigm for psychophysiological interactions. *Archives of General Psychiatry*, **24**, 221–229.

COHEN, L M (1982) A current perspective of pseudocyesis *American Journal of Psychiatry*, **139**, 1140–1144.

MILNER, G. L & HAYES, G. D. (1990) Pseudocyesis associated with folie à deux. *British Journal of Psychiatry*, **156**, 438–440

MORTIMER, A. & BANBERRY, J. (1988) Pseudocyesis preceding psychosis. *British Journal of Psychiatry*, **152**, 562–565.

TAYLOR, J. & KREEGER, A. (1987) Recurrent pseudocyesis and hypomania. *British Journal of Psychiatry*, **151**, 120–122.

Ethics of 'brain transplants'

Sir: O'Shea (*Journal*, August 1990, **157**, 302) raises the issue of brain transplants and their relation to the ancient debate concerning mind and body. A number of points seem worth making.

The hypothetical operation of transplanting A's brain into B's body (and *vice versa*) was discussed by Shoemaker (1963) and further elaborated by Williams (1970) in connection with the problem of personal identity. The issue initially appears to be whether one believes that the self (however constituted) will similarly be transferred with the brain or will remain with the body. Williams shows that both views can be cogently argued for, and discusses the links between the issue of personal identity and that of the relation of mind and brain.

It might be thought that such speculation was best left to the realms of science fiction, but there are two reasons for regarding discussion of such hypothetical cases as important. Firstly, as O'Shea implies, less dramatic forms of brain surgery already occur. One example is that of commissurotomy for intractable epilepsy. The philosophical difficulties raised by this operation are addressed by Nagel (1976). He discusses how many minds these patients can be said to have and shows that the results of the operation tend to break down our natural assumption that we (the unoperated) have one mind.

A second reason for considering these operations lies in the special way that they point up the links between issues in the philosophy of mind and ethical difficulties that are of particular relevance to psychiatry. Consider the question: How much change does someone have to undergo before thay do not exist any more (for example, removal of the brain and replacement by another)? This now looks very like the question that we ask and answer in cases of brain death. From this point it is a small step to considering cases of direct relevance to psychiatry such as the dementing relative ("she's not herself any more") or the psychotic patient during a florid episode ("he's changed beyond all recognition").

O'Shea's informal poll of a number of psychiatrists produces much the same result as my own. Most psychiatrists appear to hold either to a form of materialistic behaviourism or to some kind of dualistic position, and few are aware that the debate has moved on considerably. Neither position is philosophically coherent (good critiques of both can be found in Smith & Jones (1986)). Importantly, it cannot be a matter of indifference that we hold incoherent positions, since they do have practical consequences and lead to incoherent actions. An example worth considering in this respect would be the rationale of offering a combination of psychotherapy and medication for depressive illness. Whenever this question is discussed it is clear that (often unexamined) assumptions about the philosophy of mind are in operation.

We have begun to recognise that these issues are important. A philosophy group has now been formed within the College, and at my own hospital we now hold regular philosophy meetings.

CHESS DENMAN

Department of Psychiatry
Guy's Hospital
St Thomas Street
London SE1

References

NAGEL, T (1976) Brain bisection and the unity of consciousness In *The Philosophy of Mind* (ed. J Glover), pp 111–125 Oxford. Oxford University Press.

SHOEMAKER, S (1963) Self knowledge and self-identity New York Ithaca

SMITH, P. & JONES, O. R. (1986) *The Philosophy of Mind. an Introduction*. Cambridge: Cambridge University Press.

WILLIAMS, B. (1970) The self and the future. In *The Philosophy of Mind* (ed. J. Glover), pp 126–141. Oxford: Oxford University Press

CORRIGENDUM

Journal, August 1990, **157**, 316. The author of *Not Always on the Level* is E. J. Moran Campbell.

A HUNDRED YEARS AGO

Beer in Asylums

Dr Hearder, in the Twenty-third report of the Joint Counties' Asylum, Carmarthen, says: "It is now eight years since you ordered that the use of beer as an article of diet should be discontinued in your asylum. There was at that time only one county asylum in which the dietary did not include beer. At the present time malt liquor is included in the dietary in only a decided minority of asylums, and this number is growing smaller year by year. In this asylum the change has been decidedly and absolutely beneficial; it was not accompanied by any fraction or discontent amongst the patients. A few years previously, in 1876–7, the use of alcoholic stimulants, in the treatment of disease and as an addition to the diet of the feeble and aged, was gradually and almost completely discontinued. In this change also, there can be no doubt, we made a decided advance in rational treatment. We effected no saving in money, because more than an equal expense was incurred in providing substitutes for these alcoholic extras in the form of milk and eggs, our expenditure for eggs alone being considerably over £100 per annum. Our average annual expenditure for wine and spirits, for the 11 years ending 1876, was £90, while our average number of patients was 255. For the last 11 years, with an average population of 460, our wine and spirits bill has averaged only £12 per annum. Comparing then our mortality for these two periods, we find that during the first period of 11 years our average annual mortality was 9.7%; and during the last 11 years it has been less than 8.1%. I would not be understood to hold that this diminished mortality resulted entirely from our abstinence from alcohol, there were doubtless many other influences at work, but we may fairly claim that our patients have not suffered thereby. The disuse of alcoholic stimulants is being gradually carried out in asylums in general. In 1876 the average cost per head for 'wine, spirits and porter' was 6s. 6d; in 1886 we find it had been reduced year by year to a half of this amount."

Reference

Journal of Mental Science, **36**

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Morton Hospital, Surrey

British Journal of Psychiatry (1990), **157**, 627–629

Books Reconsidered

Bereavement: Studies of Grief in Adult Life: C. M. Parkes*

When he submitted his dissertation on *Morbid Grief Reactions: A Review of the Literature* to the University of London in 1959, Parkes made his first major contribution to the study of grief and bereavement. There followed a series of publications (e.g. Parkes, 1965, 1971) which culminated in the first edition of the book reconsidered here. On my first reading of this book, I was reminded of Samuel Johnson's comment that "the two most engaging powers of an author are to make new things familiar, and familiar things new". Parkes' descriptions of unusual reactions to loss are presented in a fashion which makes them feel reassuringly familiar, and those reactions which are more commonplace are described in a way which requires us to consider them anew.

Bereavement is not an unusual event in life; death and other forms of loss are ubiquitous features of the life style. However, until fairly recently, bereavement was not regarded as a legitimate or even attractive topic of scientific inquiry. Because of the vulnerability and sensitivities of the bereaved, many researchers have felt intimidated from pursuing the topic. Others may have felt that dealing with grief was the province of our spiritual advisors, or have feared that working in an area characterised by suffering and distress would be too daunting a challenge. However, this book offers much reassurance and confirms Parkes' own claim that "... grief, like any other aspect of human behaviour, is capable of description and study, and when studied it turns out to be as fascinating as any other psychological phenomenon" (Parkes, 1972; p. xi). One might add that, compared with some phenomena, grief is an even more attractive one because of its ubiquity and because of the opportunities there are to ease the suffering of our fellow men and women after bereavement.

When reviewing the second edition of this book in 1987, I was struck by the fact that relatively few changes had been made apart from the incorporation of about 70 more references. This, however, is not an indictment of the efforts of Parkes but rather a confirmation of the durability of his earlier findings and observations. There are now available a number of excellent comprehensive texts on this subject (e.g. Raphael, 1984; Stroebe & Stroebe, 1987), but Parkes' first book could justifiably claim to be a landmark in our exploration of the origins, nature and significance of grief.

Although psychoanalysts including Freud, Abraham and Klein, had accorded bereavement an aetiological and theoretical role in their formulations, they were not concerned with conducting a systematic study of the subject. However, a major tragedy in America – the Coconut Grove Fire Disaster – was to inspire the first such study (Lindemann, 1944). After that, there were a number of helpful descriptive accounts of the impact of bereavement, including those by Langer (1957), Maddison & Walker (1967) and Averill (1968).

Nonetheless, these early forays into the field left many questions unanswered and many issues were ignored. The relationship between 'normal' and 'pathological' grief was unclear, and there were few prescriptions (other than non-secular ones) for helping the bereaved. Such efforts did however foster a growing awareness that bereavement was a highly significant life event with important implications for the health and welfare of those who experienced it. An authoritative contribution to the topic was called for to further our understanding of the effects of bereavement, and to provide purpose and direction to the efforts of those whose job it was to help the bereaved through their period of transition. The first edition of Parkes' book, based on carefully collected data, emerged therefore as a timely and influential text.

Although he refers to some of the limited research findings available at that time, Parkes relies mainly on the information derived from the Bethlem and London studies, and from work carried out later by his American colleagues and himself in Boston. The London work involved a follow-up of 22 widows, under the age of 65 years, to find out how they would react to and cope with the loss of a spouse. In the Bethlem study, the aim was to explore the nature of atypical reactions to bereavement shown by 21 individuals (four men, 17 women). The American study involved a higher proportion of men (19 widowers, 41 widows), and aimed to determine why some individuals negotiate quite successfully the problems imposed by a bereavement whereas others do not do so and may require professional assistance. Clearly, the identification of those factors which put specific individuals at risk of adverse reactions after bereavement is a valuable contribution, particularly because it provides an opportunity for prophylactic intervention.

---

*New York: International Universities Press (1972)

Highlighted by reference to his case material, Parkes details how individuals commonly react to bereavement (and other kinds of loss such as amputation). Special attention is given to distinctive features such as pining and searching, and anger and guilt. Familiarity with such reactions is important for doctors and others who have to deal with the bereaved in order that they can reassure those whose reactions are within the normal range, and can identify those who require specialised help (the fact that such individuals are in a minority is emphasised). However, because we are social beings, it is the provision of help within conventional social settings that Parkes believes can be helpful to most of those burdened by their grief. Basic social and practical help may be provided by lay groups, such as Cruse or the Society for Compassionate Friends, but Parkes' approach to the subject confirms that we can all do something to ease the suffering of the bereaved. We can help by attending to basic practical matters on their behalf, and we can show a willingness to try to understand and share this suffering without resorting to trite comments, clichés and empty reassurances – and without being overwhelmed by their grief.

Religious convention and ceremonies, including funerals, are regarded by the author as serving a very useful function. Not only do they legitimise emotional expression and provide communal opportunities for this, but they also offer the bereaved a sense of direction and purpose. Whether the abbreviation and dilution of the previously extensive funerary rituals is in the best interests of the bereaved is questioned by Parkes, and consequently the Church is urged to assume its responsibility in providing direction and guidelines for those who are in grief.

Perhaps prescient of the concerns to be expressed later in the 1970s about benzodiazepine dependence and other consequences of regarding medication as a panacea for a person's suffering and discontents, Parkes has reservations about the extent to which psychotropic medication should be used to alleviate the pain of normal grief – although he does not deny the value of the selective use of night sedation. The problem of dependence however is not confined, as Parkes himself points out, to medication because he declares that Cruse (the organisation to which he has devoted so much of his time and energies) is "an organisation for transition rather than a perpetual refuge . . ." (Parkes, 1972; p. 177).

The book does not just contain a series of descriptive accounts or a set of prescriptions; Parkes has attempted to establish his findings and ideas within a theoretical framework. Concepts and principles, deriving from the work of Klein, the ethologists and from psychoanalysis, play a part in shaping Parkes' theoretical position. However, it is not to detract from his own work to say that it was fortuitous and invaluable that Bowlby (1969) had already laid an excellent foundation for an understanding of human relationships and of the consequences of their termination. The debt to Bowlby is certainly considerable, as Parkes himself recognises.

This is not a text, however, for the confirmed academic who has an exclusive appetite for a theoretical perspective. Nor does it provide a comprehensive review of the contemporary literature. Moreover, it is not even a practitioner's manual, providing a detailed outline of practical counselling and psychotherapeutic skills (a contribution later to be made by Worden; 1982). One could also point out that there is a biased concentration of the reactions of widows rather than widowers in Parkes' empirical studies, and it is grief and bereavement only from an adult's perspective.

Why then has this book become such an influential text, securing an unimpeachable place in the literature on grief and bereavement? I think several factors help to answer this question. Firstly, it was written by a practising clinician who has been able to combine methodological rigour with a compassionate approach to this subject. Secondly, it is written in a clear and simple style which gives it an appeal to professional and lay helper alike. Thirdly, the method of data collection was systematic and extensive, thereby giving the reader some confidence in the validity of the observational claims and findings. Fourthly, because of the relatively undeveloped nature of the field at that time, the book fostered an interest in the subject and served a propaedeutic role. It was certainly an early confirmation that Parkes was to become an outstanding teacher, researcher and clinician in this field of work. It is a fitting tribute to Parkes' contribution that his name is the most commonly indexed in two recent and influential books (Worden, 1982; Stroebe & Stroebe, 1987) and, in what is possibly the most comprehensive book on the subject (Raphael, 1984), his name is second only to Raphael's in terms of frequency of citation.

Finally, despite the inevitable pain which follows a bereavement, Parkes tries to offer hope and foster a positive attitude by regarding loss as an opportunity for change and development.

In conclusion, this first edition is a highly commendable and readable book which was welcomed by professionals and laymen who sought a compassionate but empirically justified approach to the bereaved. It is a capably researched account of the impact of and reactions to bereavement by an academic clinician, who has an unchallenged pedigree in terms of his interest in the welfare of those who have suffered the loss of a loved one. Because of this book, I suspect, many individuals will have resisted the temptation of turning away from grief: a reaction which Parkes himself indicates "will only add to the fear, perpetuate the problems, and miss an opportunity to prepare ourselves for the changes that are inevitable in a changing world" (Parkes, 1972; p. 195).

References

Averill, J. (1968) Grief: its nature and significance. *Psychological Bulletin*, **70**, 721–748

Bowlby, J (1969) *Attachment and Loss Vol 1. Attachment* New York Basic Books

Langer, M (1957) *Learning to Live as a Widow*. New York Gilbert Press

Lindemann, E (1944) Symptomatology and management of acute grief. *American Journal of Psychiatry*, **101**, 141–148

Maddison, D. C & Walker, W L. (1967) Factors affecting the outcome of conjugal bereavement *British Journal of Psychiatry*, **113**, 1057–1067

Parkes, C. M (1965) Bereavement and mental illness Part 1 A clinical study of the grief of bereaved psychiatric patients *British Journal of Medical Psychology*, **38**, 1–12.

—— (1971) Psychosocial transitions: a field for study. *Social Science and Medicine*, **5**, 101–115.

Raphael, B. (1984) *The Anatomy of Bereavement*. London. Hutchinson.

Stroebe, W. & Stroebe, M S. (1987) *Bereavement and Health*. Cambridge: Cambridge University Press

Worden, J W (1982) *Grief Counselling and Grief Therapy* London Tavistock.

David A. Alexander, *Senior Lecturer, University of Aberdeen, Department of Mental Health, Foresterhill, Aberdeen AB9 2ZD*

British Journal of Psychiatry (1990), **157**, 630–643

Book Reviews

Editor: Sidney Crown

From Neuropsychology to Mental Structure. By Tim Shallice. Cambridge Cambridge University Press. 1988. 462 pp. £15.00.

The rapid and irresistible rise of cognitive neuropsychology over the last 20 years marks a radical shift in the established methods of experimental psychology. Cognitive neuropsychologists boldy interpret their experimental results in terms of cognitive processes that cannot be observed directly. They intensively study single cases rather than groups. They believe that results from abnormal cases provide important insights into normal functioning. In part, Shallice's book is designed to convince the sceptic (probably an old-fashioned experimental psychologist) of the validity and importance of this approach. Many of us, particularly if we are students of abnormal psychology, will need little convincing. Nevertheless, we will all benefit from and enjoy following the rigorous arguments which Shallice applies to a series of case studies on dyslexia, aphasia, amnesia and many other fascinating deficits.

The cognitive approach to these disorders assumes that for normal functioning in any particular domain, several cognitive processes are required. To understand a deficit, these processes much be examined individually. The particular skill (amply demonstrated in this book) lies in specifying the processes and devising ways of separating them out experimentally. For example, reading depends (among other things) on recognising what a word means and also how to pronounce it. These two processes can be separated by contrasting the reading of non-words (e.g. 'cernalit') with the reading of words. Studies of dyslexic patients suggest that these two processes are mediated by different brain systems. In deriving predictions from these processing models of behaviour it is essential that our arguments be rigorously logical, otherwise there is the danger that we are merely playing with words. The final aim of the cognitive approach is that the models should be computational rather than verbal, hence the attraction of computer simulations such as those associated with parallel distributed processing.

For those whose primary interest is in neuropsychiatry, the last three chapters will be the most important. These concern the role of the frontal lobes and the nature of consciousness. Both of these topics are very relevant to our understanding of psychosis. The similarity in the life-styles of patients with frontal lobe lesions and those with chronic schizophrenia is striking. Much has been written on the function of the frontal lobes and most of the descriptions are vague and untestable. Shallice goes beyond this since his model (the supervisory attentional system) is derived from computational approaches. On this basis he has been able to develop new 'frontal lobe' tasks.

This book is concerned with the essential first step in linking the mind and the brain. This step requires that we can describe psychological processes in terms that will ultimately map onto brain systems. The second step is to achieve that mapping. Precisely the same approach will be needed to understand the symptoms of psychosis and to map these onto brain function.

Chris Frith, *Psychologist, Clinical Research Centre, Watford Road, Harrow, Middlesex*

Stress Disease and Evolution. By Graham W Boyd. Australia: University of Tasmania. 1989. 246 pp. Aus$29.95.

In this book, Boyd presents his ideas regarding medicine and evolution. He starts by offering his belief on how medical and scientific methodology and theory should be formulated. His views here are cogently argued and ones I have a degree of sympathy with. Boyd then goes on to expound his belief that myocardial infarction is largely due to vasospasm of the coronary arteries induced by stress. Again, this is well argued and perhaps deserves further investigation.

Following this, the author then tries to show that stress-induced vasospasm could well be responsible for numerous other medical illnesses including epilepsy, ulcerative collitis, asthma and eventually cancer. Although an interesting idea, the evidence is somewhat lacking for the role of vasospasm in these conditions.

Eventually, Boyd gives his views on evolution. He discusses a number of problems that face current evolutionary theories and believes they can be explained if evolution is seen as a process almost entirely brought about by transmission of genetic material by viruses and particles. The author then returns to his pet theory of vasospasm and believes that in higher animals this again has played a major part in the alterations of genetic material resulting in further evolutionary development.

I feel that it is very difficult for me to adequately review a book that includes so many theories and ideas from various sciences. The book ranges from philosophy through general medicine into molecular biology, genetics and evolution.

Although Boyd wishes to implicate stress as the cause of vasospasm (and therefore indirectly influencing evolution), there really are only very small portions of the book devoted to aspects of stress and psychology. This is not a book that can be recommended as important reading for psychiatrists. In the past some psychiatrists have been accused of having an interesting theory and then trying to use this theory to explain the world. This book shows that it is not just psychiatrists who perhaps fall into this trap.

CHRIS THOMAS, *Consultant Psychiatrist, Stonebow Unit, County Hospital, Hereford*

Lectures on the History of Psychiatry: The Squibb Series Edited by R. M. MURRAY and T. H. TURNER. London: Royal College of Psychiatrists. 236 pp. £10.00.

In recent years the history of psychiatry has been enjoying an explosion of activity, with a proliferation of publications on the subject, the formation of a special interest group in the College and the creation of a new journal, the *History of Psychiatry* The appearance of this book is thus timely. It brings together 12 of the annual Squibb lectures, from the series initiated in 1973. (Sadly, four of the lectures could not be published). The collection provides an interesting overview of the changing academic approaches to the subject during the last two decades. The contrast between Birley's 1974 lecture and that of Turner in 1987 is instructive. Birley's lecture draws on his wide general reading and makes no claims to be original historical research. Turner's lecture is based on extensive analysis of 19th-century case-notes and his findings are related to the latest historical scholarship. This change in approach has, no doubt, been stimulated by the work of historians who have provided a fresh and often challenging interpretation of psychiatry's past. Most of these important scholars, such as Bynum, Porter, MacDonald and Scull are represented in this volume.

Porter offers a characteristically erudite and witty study of the diary of a 17th-century madman, while Bynum provides a thoughtful essay on the relationship between psychiatry and neurology in 18th- and 19th-century Britain. Scull's "Gothic tale" relates a forgotten but alarming episode in early 20th-century American psychiatry during which large numbers of patients were subjected to major bowel surgery in the belief that this would alleviate their mental symptoms. MacDonald's lecture on insanity in early modern England examines how political and religious concerns shaped society's view of mental disorder.

A minor criticism concerns one or two historical inaccuracies in the book. Thomas Clouston worked at the Cumberland and Westmorland asylum, not the West Riding asylum, before he returned to Edinburgh (p 107), the Fife and Kinross asylum became Stratheden Hospital (p. 22).

Most of the essays in this volume have appeared before and will be familiar to those interested in the subject. Nevertheless, it is useful to have them gathered together under one cover and for those becoming interested in the history of psychiatry it provides a good introduction. It is to be hoped that further research in the field will involve not antagonism, but co-operation between historians and psychiatrists. The joint editorship of the *History of Psychiatry* by an historian and a psychiatrist is an encouraging sign for the future.

ALLAN BEVERIDGE, *Consultant Psychiatrist, West Fife District General Hospital, Dunfermline, Fife, Scotland*

The Genetics of Neuropsychiatric Diseases (Wenner-Gren International Symposium Series Vol. 51). Edited by L. WETTERBERG. Basingstoke: Macmillan Press. 1989. 363 pp. £50.00.

This is a fascinating book based on a Wenner-Gren Symposium in May 1988 at the Karolinska Institute in Stockholm. The book achieves its objectives in bringing together current research and future speculations on the new genetics of neuropsychiatry. No doubts are left about the rich progress afforded by the current vogue for linkage analysis and molecular genetic (the 'new genetics') methods for neuropsychiatric disease of known mendelian modes of transmission. The current rate of activity is so great that the book is already out of date in some aspects. However, there is no other recent compilation of papers on this subject which are of such a broad range. Therefore, this book is worthwhile for general psychiatrists as well as for the more expert reader.

The principle drawback of the book is that the wide importance of genetics as a whole and classical methods of study are rather understated.

The book has four sections. Two are on research methods: the first introduces genetic loci, genetic maps, restriction fragment length polymorphisms, linkage analysis, and chromosomal abnormalities; the second is recommended and has good chapters on phenylketonuria, fragile X syndrome and acute intermittent porphyria.

The third section is on neuropsychiatric diseases and has chapters on Huntington's chorea, myotonic dystrophy, Wilson's disease, Giles de la Tourette syndrome, affective disorders, schizophrenia, Down's and Alzheimer's disease, narcolepsy and alcoholism. Most general psychiatrists would give their eye teeth to know the whereabouts of genetic loci for affective disorder and schizophrenia. Suggestions are given: affective disorder on chromosome six or eleven or the X chromosome; schizophrenia and psychosis on either chromosome five or the sex chromosomes. Subsequent work has not consistently replicated any of these diverse results; hence the notion of aetiological heterogeneity has been invoked. Contrariwise, evidence for linkage of early-onset familial Alzheimer's disease on the proximal long arm of chromosome 21 has become stronger since publication of this book.

The fourth section, on future research, is stimulating. Brain-specific genes and homeotic genes are well discussed. The last chapter predicts that the polymerase chain reaction will prove a more and more important technique and two years later this is obviously true. The epilogue on a DNA segment on the long arm of chromosome five, thought by some to be linked to schizophrenia in some families, is of interest. Although a gene for schizophrenia looks much less likely, a much rarer condition, spinal muscular atrophy, has just been mapped in this region. The appendix on genetic loci of interest to psychiatrists is a thoughtful addition. However, the typesetting format of the book and some spelling errors are minor detractions (e.g. "corion willi" should be "chorionic villi" and "load score" should be "lod score").

In summary, this volume is fairly costly for the hard-up psychiatric researcher, but could profitably be added as a reference work in his/her local library. Certainly, the book will enlighten all who want to know more of the new genetics of neuropsychiatric disease.

MATTHEW SARGEANT, *Research Worker and Honorary Senior Registrar in Psychiatry, Department of Psychological Medicine, University of Wales College of Medicine, Cardiff*

Principles and Practice of Forensic Psychiatry. Edited by ROBERT BLUGLASS and PAUL BOWDEN. Edinburgh: Churchill Livingstone. 1990. 1584 pp. £150.

It's blue, bulky and expensive. This perhaps befits a milestone in forensic psychiatry in Great Britain, previously served only by a handful of short texts, some of them not even written by forensic psychiatrists.

Both editors are well qualified for this work. Bluglass holds a personal Chair in forensic psychiatry at Birmingham University, runs the largest medium secure unit in the country, and has been a significant lobbyist on Mental Health Law. Bowden holds one of the few remaining joint appointments, as an National Health Service consultant forensic psychiatrist at the Maudsley Hospital and a Home Office consultant to Brixton Prison. They have chosen over 140 authors from the UK, North America, Europe and Australasia, most of whom are acknowledged as experts in their subjects, although a few are not. The range of contributors includes academic and practising lawyers and social workers, sociologists and criminologists, psychologists, psychiatrists practising in each of the recognised specialties in psychiatry, a civil servant, a statistician and a probation officer.

The text is divided into 15 sections which cover law, crime, the justice process, administration, services and the role of psychiatric disorder in offending, for adults, children and adolescents. It is reflective of society and the criminal justice system in focusing almost exclusively on the offender, with just 25 out of 1405 text pages reserved for victims. There is an excellent table of relevant legal cases, but an unfortunate omission is in the failure to list Government documents. The reference list is extensive and the index imperfect, but far better than most. A fascinating touch is provided by the 22 plates, of the bad and mad, the illustrious and some great institutions.

Advantages to the editorial style are undoubted in the breadth of information and views which have been gathered together. There are some eccentricities, but these tend to provide a levening influence. The section on institutions and services, for example, does provide the expected chapters on the probation service and maximum and medium security in hospitals, although there is nothing from Gunn on prisons, not even a reference to the first chapter of Gunn *et al's* book *Psychiatric Aspects of Imprisonment*, in the section on the history of the prisons. The remaining contributions, however, imply friendship rather than theme, with entertaining chapters about a prison or two in Britain, elsewhere in Europe, a Dutch health facility for psychopaths and Danish forensic psychiatry services.

The disadvantages of the light editorial style are considerable. A significant problem is the tremendous repetition. In section IV, for example, on "The mental element in crime", chapters one and two contain so much overlap that the reader can spare effort and focus on the first as the more encompassing. In addition, the essentials of chapter five, on automatism, are well covered in three and four, and the chapter is also one example of a number of major imbalances in the book. In case terms, automatism is numerically insignificant, but, with the chapter on hypoglycaemia, more than 20 pages are thus devoted to it. The comparable allowance for all the other aspects of mental responsibility in English Law, including the repetition, is 33 pages, with just six each for Scotland and for the USA, and seven for Europe.

The main purpose of this book must be as a reference volume. Its organisation makes this difficult at times. Do not, for example, look in the section on "Law and psychiatry" for Mental Health Law. That comes under "Legislation", some 11 sections and more than a 1000 pages away. Those needing advice on being an expert witness through the criminal justice system must sample extensively from at least each of sections I, III, IV and VII and VIII as well if their patient is not of majority. This seems a book primarily for senior practitioners.

Overall this is not a text for reading, although individual chapters will bring their own reward. It should be a useful source book. Ironically, as a textbook of forensic psychiatry edited by psychiatrists, it is the sections on law that are strongest. This is true even of the sections on child and adolescent law, which suffer an inevitable problem in a ever-changing field: that they had to anticipate the 1989 Children's Act through preceding bills rather than document the Act *per se.* I will certainly dip into the book and use the reference list. Should each have their own copy? That depends on how you want to spend the next £150!

PAMELA J. TAYLOR, *Head of Medical Services, Honorary Senior Lecturer, Institute of Psychiatry, London*

Confidence Interval Analysis: Manual and Disk. Edited by MARTIN J. GARDNER, STEPHEN B GARDNER and PAUL WINTER. London: BMJ. 1989. 77 pp & disk. £65 or £45 NHS.

Statisticians are often heard to complain about the over-emphasis placed on significance testing by researchers in medicine and related disciplines, which frequently results in the literature of these subjects being liberally scattered with *P* values or 'stars' to indicate particular significance levels. In recent years such complaints have been taken to heart by editors of journals such as the *British Medical Journal* and the *Lancet,* who now expect scientific papers submitted to them to contain, where appropriate, confidence intervals rather than simply the presentation of a *P* value from a hypothesis testing procedure. This change of emphasis is to be applauded since in medical studies investigators *should* usually be interested in determining, for example, the size of difference of a measured outcome between groups, rather than a simple indication of whether or not it is statistically significant. Confidence intervals present a range of values, on the basis of the sample data, in which the population value for such a difference may lie.

The computer program Confidence Interval Analysis (CIA), designed for IBM-compatible microcomputers, should encourage the change from significance testing to the calculation of confidence intervals, since it is extremely easy to use even for that ubiquitous character, the research worker who is naive where statistics are concerned. The CIA is menu driven and can be used to provide confidence intervals for means, regression slopes, odds ratios, hazard functions, etc. Either raw data or summary statistics may be entered into CIA and the options of 90, 95 or 99 per cent confidence intervals are provided. Data entry and error correcting are straightforward, and warnings are given when the confidence interval method chosen is inappropriate, for example, because of small sample sizes.

The program is meant to be used with the associated book, *Statistics With Confidence,* and together they should certainly provide the medical researcher with the tools needed to calculate confidence intervals in many circumstances of interest. In the long run this will, hopefully, lead to a further decline in those tables in medical journals which are so covered with 'stars' that they resemble a hotel guide book.

B. S. EVERITT, *Professor of Behavioural Statistics, Biometrics Unit, Institute of Psychiatry, London*

Hospital Closure. By NANCY CORMAN and HOWARD GLENNERSTER. Milton Keynes: Open University Press 1990. 173 pp. £9.99.

This book details the administrative, financial and political issues involved in the closure of Darenth Park Hospital, a large mental-handicap hospital which served almost half of the South-East Thames region. It makes interesting reading, possibly because the method resembles investigative journalism, albeit in a minor key.

The closure occupied the major part of the last decade. In this respect the book represents a splendid obituary for a style of managing affairs that already seems forgotten. For this reason it has an historical importance but not necessarily much relevance for the 'great leap forward' we are now embarked upon.

The clinical issues arising are dealt with in *Hospital Closure and the Resettlement of Residents* by Lorna Wing (1989, London: Gower Publishing)

GREG WILKINSON, *Director, Academic Sub-Department of Psychological Medicine in North Wales, North Wales Hospital, Denbigh, Clwyd*

When One Wants Out and The Other Doesn't: Doing Therapy with Polarised Couples. Edited by JOHN F CROSBY. New York: Brunner/Mazel. 1989 240 pp $41.00.

The type of marriages focused on in this collection of 12 clinical essays are accurately referred to by the editor as "polarised marriages": those couples who present with one partner wanting to finish the relationship and the other wishing to continue it The editor describes these as "non-pathological marriages", suggesting that they may have become "devitalised" and contrasting such couples with those who might present for help with a more specific symptom or problem, such as a psychiatric condition, an addiction, or an affair. What is surprising is that the extreme polarisation is not itself recognised by the editor as being the symptom.

Each partner in a marriage inevitably has to cope with some degree of ambivalence towards the differences and different needs of the other partner, and also has to deal with tensions between independence and dependence. When the partners lose their capacity to carry this ambivalence within themselves, extreme splitting may occur, and the marriage may become polarised and rigid, with each partner carrying one side of the ambivalence. The marriage may then be said to be in an impasse.

In clinical practice, such a presentation is not unusual, and the couple have to be understood, be it systemically or psychodynamically, as manifesting *between them* the two sides of the ambivalence about their relationship. Therapeutic practice needs to include an understanding of the unconscious processes of splitting and projection, which can sometimes be very powerful. It is because of this powerful mental mechanism that such couples are rarely 'devitalised'. Usually, the opposite is the case, with the polarised feelings resulting in anger, anxiety and sometimes paranoia. Many of the papers in this volume describe how difficult it is to work with such couples because of the constant pressure to side with either one or other of the polarised partners. It may be said that unconsciously the therapist is being pulled into either one or other side of the split and is not being allowed to relate to both sides.

The theoretical orientation of the authors of this volume is eclectic. One writer states that his approach includes "elements of psychoanalysis, social learning theory and behaviourism mixed in with systems thinking". Such a theoretical mix may allow the marital therapist to make a broad diagnostic assessment, but it leaves the problem as to how to intervene clinically. The evidence from the authors is that they took a cognitive systemic approach, and paid very little attention to unconscious processes, either in the marital interaction or in the psychotherapeutic encounter. This was the case even with one author who outlined psychoanalytic object relations theory as her theoretical orientation and then proceeded to describe a cognitive and pragmatic treatment model.

This reviewer found the book disappointing and at times confused. The clinical population chosen for consideration is a very challenging one, with the powerful splitting often making psychotherapeutic work slow and difficult. With splitting being the major dynamic in the clinical work, a unitary conceptual and clinical orientation would be indicated, rather than a more eclectic one which might more easily become subject to the pushes and pulls of the underlying dynamic.

STAN RUSZCZYNSKI, *Senior Marital Psychotherapist,*
Tavistock Institute of Marital Studies,
Tavistock Centre, London

Suicide: Understanding and Responding. Edited by DOUGLAS JACOBS and HERBERT N. BROWN. USA: International Universities Press. 1989. 505 pp. $60.00.

In the era of 'new eclecticism', where the American language is the norm and many no longer blanch at a phrase such as 'the patient then suicided', there is something of interest in this book for most. The extent of American isolationism is apparent in the absence of much European literature on this vast subject, with the exception of the more biological aspects in which the Scandinavians reign supreme with, of course, Peter Sainsbury. European Literature (with a capital 'L'), however, *is* in evidence, the authors of the papers being a generally well read bunch, and thus adorning chapters on suicide with reference to figures like Sylvia Plath. A similar compendium from a London teaching hospital could contain mountains on Virginia Woolf, I suppose. However, I cannot think of a teaching hospital or institution in this country which could mount a comparable list of authors of such depth and experience on one major subject.

This book indulges in a fair amount of transatlantic psychospeak, and a number of very differing styles, from an informal "given that I am a psychologist . . ." one to a much more rigorous academic style. Surprisingly, this variance gives the book a high readability quotient, much like a *Reader's Digest* anthology of filleted classics. The book is divided into "Foundations", "Understanding", "Responding" and "Philosophical perspectives", with a wide range of chapters on treatments including those which are physical, broadly psychotherapeutic, and more specifically post-Freudian. There are also chapters on the nature of assessment and the hospital milieu, as well as sensible articles on dealing with in-patient suicides from a staff support point of view. The verbatim sample interview is less interesting, however, as is the chapter on "The empathic method of psychotherapy with suicidal patients".

Szasz's chapter entitled "A moral view on suicide" is worth the not inconsiderable purchase price on its own. He suggests that: "The claims of psychiatrists seeking an understanding of suicidal behaviours are thus every bit as absurd as would be the claims of right-to-lifers (who call abortion 'murder of the foetus') that they are scientists seeking an understanding of foeticidal behaviour". Elegant, but what does it mean? And, inevitably: ". . . there is neither philosophical nor empirical support for viewing suicide as different, in principle, from other acts, such as getting married or divorced, working on the Sabbath, eating shrimps, or smoking tobacco"; and "the fact the psychiatrist plays such a decisive and distinctive role in the contemporary politics and practices of suicidal prevention points to a hidden agenda, namely, to our general reliance on psychiatric explanation of disturbing behaviour and on psychiatric control of disturbing persons". Szasz raises the issue of the 'psychiatric will' which was first espoused in 1982. This is meant to encourage all citizens to establish their wishes clearly, and legally, should they be deemed to require coercive psychiatric methods of suicide prevention, thus stopping a patient "from being coercively paternalised by psychiatrists"

Buy this book for the local psychiatric library, and many of its multi-authored chapters will give food for thought and topics for discussion.

DAVID ROY, *Consultant Psychiatrist,*
Goodmayes Hospital, Barley Lane, Ilford, Essex

The Ailment and Other Psychoanalytic Essays. By TOM MAIN. London: Free Association Books. 1989. 280 pp. £27.50.

Psychoanalysis as applied to psychiatric practice has followed a very special tradition in the United Kingdom. In this, the psychoanalyst, using his/her particular training and skills, has focused his/her interest on the total hospital environment as a means of helping the in-patient to recover. Such a concept of the mental hospital as a therapeutic community owes its beginnings to the pioneering work of a number of psychoanalysts at Northfield Hospital at the end of the last World War. This experiment, in turn, led to another one at the Cassel Hospital where Main became Director in 1946 and continued in this role until 1976.

Much of this collection of essays and speeches is devoted to his work at the Cassel Hospital. They are introduced by Rayner and his daughter, Johns, who edited the book. Its title is appropriate, for it is *The Ailment* which has been standard reading for psychiatrists in training for many years. This paper provides an outstanding discussion of that group of 'special', rather masochistic patients who can command so much attention from their therapeutic environment with so little by way of reward. This and other of Main's essays are devoted to studies of the therapeutic community and its implications for helping in-patients psychotherapeutically. They give an excellent picture of the work being done at the Cassel: the hallmark of this work has always been a lively curiosity "about the external and internal significance for the patient", of his/her entry into and departure from the hospital. Regrettably, we seem to live in an age of rapid patient transit when few psychiatrists feel that they are given enough time to study these issues, let alone to look after their patients in hospital. Main and the Cassel have been so unusual in their concern to retain time for such study.

Although most of the essays are devoted to Main's interest in the application of psychoanalysis to psychiatry, three essays are written about a different set of problems concerning husbands and wives, motherhood, and a special group of patients at the Cassel, namely mothers and children. For many years Main worked with doctors in training at the Family Planning Association helping them to deal with psychosexual problems. This led to the foundation of the Institute of Psychosexual Medicine, of which he is Life President. His essay entitled "A fragment of mothering" throws an interesting light on the mother in her dual relationship to her child and her husband, and explores her conflicts in this situation; it places more emphasis on aspects of the mother's sexuality than other psychoanalytic writers have done.

Some of the essays in this book are repetitious in their themes. I would have preferred a more conservative selection. This book comes at a timely moment, when the Cassel Hospital is under the threat of closure, and provides a valuable historical commentary on the therapeutic community movement as it developed within that hospital. Main's optimism is a much needed antedote to a prevailing mood of cynicism and despair about the value of in-patient psychotherapy in the care of the severely mentally sick.

PETER SHOENBERG, *Consultant Psychotherapist, Joint Department of Psychotherapy, University College and Middlesex Hospitals, London*

The Birth of the Family. An Empirical Inquiry. By JERRY M. LEWIS. New York: Brunner/Mazel. 1990. 224 pp. $26.95.

Does the way in which couples deal with the formation of their own relationship predict how successfully their child will be incorporated into the family? What impact does the parents' relationship with their family of origin have in this process? How does parental psychological health affect the transition to parenthood as a major stage in family development?

In this book, Lewis describes the research into these issues carried out at the Timberlawn Psychiatric Research Foundation in Dallas over the past 20 years. They studied 38 white, middle-class couples with a stable marital history who gave birth to infants considered to be normal and healthy. They interviewed the parents, recorded interactive scenes on video before and after the birth of the child, and devised measures to assess the marital relationship and parenting in the first year of the child's life.

The results give some validity to the authors' classification of marital relationships into 'highly competent', 'competent but pained' (in that there is conflict around the issues of closeness and intimacy), 'dominant–submissive', 'conflicted' and 'severely dysfunctional'. Against hypothesis, the relatively well functioning families (the competent but pained) proved the most vulnerable to regressive change in the marriage whereas the dominant–submissive group demonstrated stability of marital structure across the transition to parenthood. The more dysfunctional marriages showed a mixed response to parenthood with both improvements and deteriorations in the marriage. Variables such as the spouse's psychological health and the perceived stress and supports from social networks had relatively little influence when compared with the marital structure itself.

Pre-natal ratings of marital competence predicted measures of parenthood such as the mother's warmth towards the three-month old baby. Interestingly, there were indications that first-born girls, but not boys, of mothers in dysfunctional marriages may be particularly vulnerable to problems in development, as judged by less investment in parenting and less adequate attachments at one year in this group. There were similarly interesting results as regards families of origin: for example, the more positively wives recorded their fathers as being during their childhood, the less competent the qualities of their marriage.

It is obvious that a lot of care was taken to devise and assess the measures of marital relationships and parenting and it is helpful to have them outlined in the appendix. Similar care was taken in the analysis of the results. However, the discussion is disappointing in that it tends to reiterate the results, and the conclusions are, of course, based on quite a small sample.

Nonetheless, this book describes research which advances knowledge in the area. It provides a valid way of classifying marriages, it shows the changes in family structure with the onset of parenting, and it describes interesting associations between child and parent gender, and between the influence of family of origin and both parenting and marital relationships. This appears to be a helpful model for studying marriage and parenting, and the book can be recommended for those interested in this area.

Elena Garralda, *Professor of Child & Adolescent Psychiatry, St Mary's Hospital Medical School, University of London*

Cognitive-Behavioural Marital Therapy. Edited by Donald H. Baucom and Norman Epstein. New York: Brunner/Mazel. 1990. 496 pp. $47.95.

Cognitive therapy is now well established as a psychological treatment for individuals with a wide variety of emotional disorders. Systematic studies of its use with couples or families is, however, relatively lacking. This book is therefore very timely. The authors are experienced and respected therapists who, having collaborated clinically to good effect, have together written a comprehensive textbook on cognitive-behavioural approaches to marital problems. This will obviously appeal to cognitive therapists who wish to develop skills in this area, but also to marital therapists who are looking for new methods of structuring their interventions.

The book is divided into three clear sections. The first deals with the theoretical aspects of this approach, the second with assessment procedures, and the final part looks specifically at therapeutic interventions. The apparent artificial division between behavioural, cognitive, and affective chapters seems to work well in practice, as each area is explored in some depth. As well as case material, the text is supplemented by clinical scales and questionnaires that will be of use in this type of therapy. I was particularly impressed by the section on how to integrate therapeutic interventions. The authors explore in some detail which intervention to use, when and why to use it and when not to. This type of process analysis is rarely presented with such clarity and will be appreciated by many.

Although this text draws on theoretical and research knowledge, it is primarily a handbook for clinicians. It makes assumptions about the therapists' general skills and level of competence. It is not written for novices. The hardback version of this text will not be cheap to purchase, but Baucom & Epstein have produced an excellent book which will be of interest to a multidisciplinary audience working with marital problems. They have set a high standard which future contributions in this area will find hard to better.

Jan Scott, *Consultant and Senior Lecturer, University Department of Psychiatry, Newcastle upon Tyne*

The Significance of Infant Observational Research for Clinical Work with Children, Adolescents and Adults. By Scott Dowling and Arnold Rothstein. Madison: International Universities Press. 1989. 257 pp. $32.50.

This book reports contributions to a workshop sponsored by the American Psychoanalytic Association and contains differing views of psychoanalysts about the impact of systematic infant-observation research on psychoanalytic theory and practice. The first of the book's three sections is a chapter reviewing what is seen to be the two main approaches to infant research: the psychoanalytic and that originating from academic psychology. The second section includes a series of workshop papers in which individual psychoanalysts describe the way that infant-observation research has influenced them. In almost every case the authors use clinical examples to exemplify their points. In the third section, the workshop papers are discussed by five different writers, including one of the editors.

The book tends to be much stronger on opinions, theory and descriptive material from individual cases than data from academic developmental psychologists. The developmental psychologists most often referred to are Stern and Emde and there is surprisingly little reference to Bowlby and work deriving from attachment theory. There is also no acknowledgement of the increasing body of empirical work tracing continuities and discontinuities in development, although the existence of these is emphasised by several authors. The psychoanalytic practitioners and theorists most commonly referred to are Anna Freud, Mahler, Kleim and Winnicott.

For those who are looking for change and flexibility in the orientation, theorising and practice of psychoanalysts there will be both encouragement and frustration in reading this book. Some of these writers see the need for major change in psychoanalytic theory, particularly in the area of drive theory and theories connected with the emergence of self–other differentiation. There are some who suggest that early infant-observation research points to the need to change psychoanalytic practice for at least some patients so that the analysis happens face-to-face and with the therapist in a more active role.

There are also a number of authors who consider the research has promoted conceptualisation of 'model scenes' of infancy, which develop in the pre-oedipal and, indeed, pre-verbal phase of development. These are probably conceptually similar to, or overlapping with, Bowlby's working models of relationships

However, there are others who see no implications for either theory or practice, including those who maintain that any insights derived from the observation research have already been discovered by psychoanalysts themselves In consequence, readers with a psychoanalytic viewpoint will find at least something to accord with their own view whatever it may be. Optimistically, this book gives evidence of increasing dialogue and rapprochement between those working within psychoanalytic and academic developmental psychological traditions.

A. D. Cox, *Professor of Child and Adolescent Psychiatry, Bloomfield Clinic, Guy's Hospital, London*

The Child in Our Times: Studies in the Development of Resiliency. Edited by Timothy Dugan and Robert Coles. New York. Brunner/Mazel. 1989. 240 pp. $25.00.

It has long been recognised that children differ markedly in their response to stress and adversity, and in recent times the concept of resilience has become fashionable. Coles defines the phenomenon as the ability to 'keep trucking' in spite of vicissitudes, and this volume comprises a series of essays on the topic. They vary in style and focus, with accounts of normal toddlers' responses to moral conflict, street children, children of alcoholics, diabetics, temperamental adversity, and escape from the 'underclass'. A few of the chapters make reference to empirical data, but the main emphasis is on clinical cases and general concepts. It is clear, of course, that resilience is not a unitary feature. It is biologically unsound to suppose that precisely the same characteristics will foster adaptability in all circumstances and, equally, it is evident that resilience will often reside in patterns of person–environment interaction over time rather than in some fixed personality trait.

This volume provides some useful ideas on a clinically, as well as theoretically, important issue, but the book would have been much stronger if the editors had provided some kind of integrating overview. As it is, most readers (or at least this reviewer!) will struggle to sort out what messages they should take away.

Michael Rutter, *Professor of Child Psychiatry, MRC Child Psychiatry Unit, London*

Childhood Illness: The Psychosomatic Approach. By B Lask and A. Fosson. Chichester: John Wiley & Sons 1989. 156 pp. £11 95.

The prologue begins with the quotation: "The psychosomatic approach is listening to the child talking with his body" (anon). This sets the scene and economically describes the theme portrayed by the authors. They are a well known British child psychiatrist and a Professor of Pediatrics, Behavioural Science and Psychiatry at the University of Kentucky Each has an outstanding reputation and I picked up this book with high expectations which were not disappointed.

There are 10 chapters. Each one begins with a quotation and several sentences of guidance to readers and finishes with a summary. The text is laid out in a hierarchy of titled and numbered paragraphs in a standard format. There are seven pages of up-to-date references. The first chapter reviews the origins, meaning and use of the term 'psychosomatic', outlines the psychosomatic approach, and establishes the basis on which interacting influences are considered in the following chapters through describing predisposing, precipitating, perpetuating and protective factors. Psychophysiological mechanisms, a review of common syndromes and the illness network, which are covered in following chapters, further elaborate the authors' systemic theme and clinical approach.

Subsequent chapters summarise the implications of the authors' clinical style for assessment and treatment, as well as the basic principles of helping parents. The last three chapters review family therapy, individual therapies and pharmacotherapy.

The style of this volume is brief and concise. Complex interacting factors are illustrated by systems diagrams, and the practical orientation of the book to clinical practice is brought alive by frequent inclusion of case vignettes. The authors state their intention as that of integration – of body and mind and of paediatrics and psychological medicine. I believe that they achieve this objective.

While I am sure that I like this book because I find myself in tune with its philosophy, I am equally certain that this is but one reason. This book is deceptively basic and, although only a slim volume, it stimulated me into thinking again about the issues involved in a demanding yet rewarding arena of practice. The authors' well ordered approach is helpful, as are the references. This book is appropriate reading for many of the professionals who work with sick children. I would particularly recommend it to medical undergraduates and believe that doctors at many different levels of postgraduate training and experience in paediatrics and psychiatry will find it useful too.

RICHARD WILLIAMS, *Consultant Child and Adolescent Psychiatrist, Department of Family Psychiatry, Bristol Royal Hospital for Sick Children*

Anxiety Disorders in Children. By RACHEL G. KLEIN and CYNTHIA G. LAST. London: Sage Publications. 1989. 140 pp. £16.75.

This is a concise book about a topic which is not often considered in its own right. Clinicians working with children are aware of how often children and their families who seek medical and psychiatric help are very anxious, but perhaps anxiety disorders *per se* are under-diagnosed.

The book starts with an excellent review of the concept of anxiety over the past century, with consideration of several viewpoints and theoretical positions. There is then a good discussion of diagnosis, especially of using DSM-III criteria to distinguish three types of anxiety disorder: separation anxiety, avoidant disorder and over-anxious disorder. This includes a detailed review of the use of standardised questionnaires for assessment. The authors reveal their recent research in this area, based on extensive clinical knowlege.

The chapter on treatment is rather brief, and concentrates on behavioural treatments, claiming that research in other (psychotherapeutic) fields is inadequate. The authors do draw on their own considerable clinical experience, yet this was the most disappointing part of the book. They briefly consider medication such as antidepressants, which they consider to be of limited usefulness in obsessive-compulsive neurosis.

A detailed review of risk factors and criticism of various studies show an aggregation of anxiety-disordered persons within biological families, without causal links being made. Stressful life events, neurological symptoms, and normal developmental fears are mentioned.

Finally, the authors consider the continuity of adult to child anxiety disorder which they consider to be of great importance in the early recognition and possible preventive treatment of these disabling conditions. They make the point that anxiety disorders in adults are very common (about 15% of population) and that they often start in childhood or adolescence.

In summary, this book provides a good review of the available literature on assessment, diagnosis and natural history of anxiety disorders in childhood. Its coverage of treatment methods is weaker and therefore less useful from a clinical point of view. It should be read by those wishing to consider the subject in depth, or undertake research in this topic.

BEVERLEY NORTON, *Senior Registrar, Royal Hospital for Sick Children, Edinburgh*

Residential and Inpatient Treatment of Children and Adolescents. Edited by ROBERT D. LYMAN, STEVEN PRENTICE-DUNN and STEWART GABEL. New York: Plenum Publishing Company. 1989. 389 pp. $50.00.

The first section of this multi-authored book deals with general issues concerning the residential treatment of disturbed children. The second reviews various therapeutic models: psychoanalytic, behavioural, 'medical', peer culture, psychoeducational and 'wilderness challenge'. Components of the 'medical' model are milieu therapy, psychotherapy, family therapy, pharmacotherapy and the remediation of language problems.

The third section starts by discussing out-patient treatment as an alternative which usually needs to be considered, and then turns to various treatment methods which may be used as part of residential programmes. These are pharmacotherapy, individual and group therapy, the involvement of parents, and educational programming. There is also a chapter on the evaluation of programmes.

The final section deals with the treatment of 'special populations' – children and adolescents who are schizophrenic, mentally retarded, conduct disordered, abused and/or neglected, and suicidal. There is also a chapter on adolescent alcohol and substance abuse. The chapter on conduct-disordered children and adolescents is unfortunately confined to a discussion of the treatment of juvenile delinquents. Not every child with a conduct disorder becomes involved with the juvenile justice system before presenting for treatment, a point of which this book fails to take note. Another problem is that there is not much on autistic children, despite the statement, in the section on pharmacotherapy, that "the number of autistic children admitted into specialised residential treatment increases as the children grow. . . ."

For the most part, this book describes practice in the United States, although there is one chapter by Canadian authors. Much of the content is, however, of wider application. On the whole, it provides a sound and up-to-date account of the practice, place and limitations of residential treatment for disturbed children. A commendable feature is its emphasis on the evaluation of programmes. This has been an area of weakness, but the authors keep us aware of this and review what is known of the efficacy of the treatment programmes.

PHILIP BARKER, *Professor of Psychiatry, University of Calgary, Canada*

Perspectives on Behavioural Inhibition. Edited by J. Steven Reznick. Chicago & London: University of Chicago Press. 1989. 315 pp. £26.25.

Two mothers and their 30-month-old children, who have never met, enter a large room with toys scattered on the rug. One girl immediately leaves her mother, walks over to the toys, and starts to play. The other girl sits close to her mother quietly watching the other child. The exploring child may talk to her mother or offer the quiet girl a toy and will spend little time next to her mother. The second girl will remain next to her mother for 10 to 15 minutes before moving to the toys and is unlikely to speak or to approach the other girl for the rest of the 30-minute session. The scenario captures the contrasting behaviour of the classic 'inhibited and uninhibited' children who form the subject matter of this book.

Behavioural inhibition refers to the child's initial behavioural reactions to unfamiliar people, objects, and contexts or challenging situations. Some children become quiet and restrained, assessing the situation and their resources before acting, while others act much more spontaneously. 'Inhibition' is often displayed as shyness in children and can be observed very early in infancy. This volume attempts to cover the current theoretical and research findings from a variety of perspectives relating to behavioural inhibition, following a conference held in Massachusetts in 1986.

Several chapters present and discuss data from the follow-up of two longitudinal cohorts of children selected to be either extreme in their initial timidity to the unfamiliar or extreme in their lack of timidity at 21 or 31 months of age. Other chapters are devoted to the discussion of comparative data using animal models or current work on elucidating the possible physiological systems that underlie behavioural inhibition. The concluding chapters concentrate on considering the phenomena in relation to clinical syndromes, such as emotional disorder in childhood, but also as a possible precursor for adult anxiety disorder.

The diversity of approach and clear presentation of research data are two strengths of this book. It is surprisingly readable, and as a clinician I welcomed the format of linking current theoretical viewpoints with possible future applications for clinical practice. Most thought-provoking and enjoyable.

Marian Perkins, *Senior Registrar Children's Department, Maudsley Hospital, London*

Clinical and Diagnostic Interviewing. Edited by Robert J. Craig. New Jersey: Aronson. 1989. 355 pp. $40.00.

This is the first time that an attempt has been made to gather into one volume the multivarious aspects of clinical interviewing. It was therefore with a sense of anticipation that I began to read. Alas, it did not fulfil its promise. Although some of the contributions were excellent, the chapters on assessment of personality and on suicide intent being especially clear, the standards set in these chapters were not maintained and the book was mostly unstimulating and prosaic.

By and large there was little new information which a trainee working in a teaching hospital would not already be familiar with. For example, the chapters on alcohol and drug abuse outlined the standard interview for assessing these conditions. The more complex aspects of assessing motivation or insight were not touched upon. In general there was failure to address the complexity of interviewing such as dealing with sensitive areas, overtalkativeness, resistance. Many of the chapters devoted too much space to outlining the diagnostic criteria and some of them devoted more than half the space to this. This book therefore frequently became a textbook.

Stylistically the chapters frequently changed half-way through. In particular, some adopted a formal approach initially and drifted into a conversational mode.

The basis of interviewing techniques such as styles of questioning and the physical aspects of talking with patients were barely touched upon in the introductory chapter and should have been given a lot more space. I am not a British chauvinist but I was disappointed that the contributors failed to mention the very valuable work on interviewing pioneered by such people as Goldberg (in adult's psychiatry), Rutter (in child psychiatry) and Tyrer (on personality assessment). This book frequently instructed the reader in what to look for during interviews but gave no guidance on how to do this. This was especially pertinent to the section dealing with psychodynamic and behavioural interviews.

Finally, I feel that it was a serious omission not to devote a chapter to research and structured interviews, although they were mentioned briefly by some contributors. While I enjoyed parts of this book, I cannot recommend it with any enthusiasm.

Patricia Casey, *Senior Lecturer, Department of Psychiatry, Regional Hospital, Cork*

Psychiatric Interviewing: a Primer (2nd edn). By Robert Leon. New York: Elsevier Science Publishing. 1989 193 pp. £17.24.

When did you last read a good book about interviewing technique? It is remarkable how few there are. This book is excellent and very readable. Many students and trainees will find it useful and interesting.

Professor Leon recommends a fairly non-directive interviewing style, believing that this produces just as much essential information. In addition, patients tend to be more satisfied with the interview if they are given the freedom to talk about what they feel is important. A directive style produces more negative findings and less spontaneous information.

Interviewers make predictable mistakes, and qualified doctors sometimes make the same ones as students. Leon is tactful in that he uses clear examples of faults taken from student interviews. For example, a common error is to ask for facts and ignore feelings.

Patient: I sometimes feel a little overprotected.
Student: Do you have any problems with your heart now?

"Starting the relationship" is a very good chapter, full of useful hints. No knowledge is assumed. "To begin, address the patient by name, introduce yourself, and state the purpose of the meeting. Except for special situations, use the patient's surname" Do not say "Hi Mary". The patient should be put at ease, if possible by some meaningful comment such as "It must be hard getting here in all that rain". The aim is then to go through the whole interview without disrupting the patient's thoughts and feelings. Various facilitating techniques can be used, for example taking a key word used by the patient and repeating it back to him.

Patient: Everyone needs a rubber duck you know, something to cling on to.
Doctor. What's your rubber duck?

It has been said that the main use of interpretations is to stop the therapist falling asleep. Leon has other ideas. Interpretations should not be given immediately as they may be only partly right and they may stop the patient talking However, they can be used to help the therapist encourage the patient towards fruitful lines of thought.

Four chapters were written by other authors. One chapter is a pedestrian account of the mental state examination. Another is on cross-cultural difficulties and it contains some useful ideas. Martinez, Jnr, mentions that the easiest people to interview are those most like ourselves.

I enjoyed this book and found I was still learning from it even when I re-read it twice. It is a conveniently sized paperback at a realistic price

If you are a despairing clinical tutor or lecturer wanting to know what to recommend, or if you are a trainee wondering how you will ever pass the membership examination, start here!

FLAVIA M LESLIE, *Senior Registrar, The London Hospital (St Clement's), London*

Health Measurement Scales: A Practical Guide to their Development and Use. By DAVID L. STREINER and GEOFFREY NORMAN. Oxford: Oxford University Press. 1989. 182 pp. £25.00

In many areas of clinical research there is a growing awareness that 'quality of life' and general health assessment comprise an important aspect of the assessment of new treatments. For example, treatments for many forms of cancer (my particular interest) frequently offer limited survival advantage to the patients, yet are accompanied by various forms of severe toxicity. The clinical trial investigators are therefore interested in assessing the difference in quality of life between the treatment groups, as well as providing information which may help physicians to decide the appropriate treatment for future individual patients. As a consequence many health measurement scales have been developed over the last couple of decades

This book, which is aimed at clinical researchers, discusses the development and assessment of such scales. It fills a notable gap in the literature, and is exactly the sort of book that I would have liked to have read when I first encountered this subject. As such, I would have little hesitation in recommending it to clinical investigators who are embarking upon the development of new questionnaires or tools for health measurement. It should also be helpful to anyone examining existing scales, trying to select one to use.

The chapter titles suitably describe the coverage of the book: basic concepts; devising the items; scaling responses; selecting the items; biases in responding; from items to scales; reliability; generalisability; validity; measuring change; latent trait theory; and methods of administration. Although statistical concepts are necessary for many topics, the authors carefully avoid the use of too much mathematics or statistics, and rely upon references for those who require a deeper understanding of the techniques. They also avoid detailed discussion of particular scales, although in many ways this is a serious disadvantage: there are too few specific examples of the application of the assessment techniques, and many of the examples provided are for fictitious or hypothetical data. When discussing validity, say, it would be useful to know whether many of the better known scales have assessed validity, how they did it, and what sort of answers they obtained In the absence of such details the reader is left with a rather abstract concept and without a feel for how important validity is in practice, and what is an acceptable level of a validity assessment. Also, on the few occasions that real scales have been used for examples, they hardly ever chose a scale that anyone in cancer or most other general fields of clinical medicine is likely to be familiar with! I was left wondering what areas of clinical research or health sciences the authors had in mind.

Although 'quantity of life' and 'quality of life' are mentioned, there is no discussion of the various attempts to assess 'worthwhileness' of treatment in a global sense, which requires a means of integrating the two dimensions. Although personally I would quesion such approaches as QALYs (quality adjusted life years), one must recognise that ultimately a doctor is interested in whether or not to administer a new treatment, and this calls for some sort of summary measure.

Finally, one important point which I feel is severely misleading in several places in the text is the use of correlation. Correlation coefficients have been widely misused for assessing agreement of two scales and repeatability; Bland & Altman, for example, in an excellent paper (*Lancet*, 1986, *i*, 307–310), cite five reasons for a high correlation *not* indicating that two methods agree. I regret that this book may encourage such deplorable misuse This is particularly regrettable in that the author is clearly aware of some of the problems and in a late chapter makes cursory reference to "biases in validity assessments", but without any suggestions of alternative methods.

In summary, although I think some aspects of this book could be greatly improved, and although I disagree totally with the implicit endorsement of misuse of correlations, I nonetheless feel that it provides a very comprehensive and readable introduction to assessment of health measurement scales. I would certainly recommend it to anyone who requires an introduction to this field.

PETER FAYERS, *Statistician, Medical Research Council Cancer Trials Office, Cambridge*

Self Assessment for Psychiatry Examinations (for the New MRCPsych Part II). By BASANT K. PURI and GREG J. WEPPNER. Edinburgh: Churchill Livingstone. 1989. 144 pp. £9.95.

This book offers candidates for the Part II examination 300 multiple-choice questions (MCQs) and 60 short-answer questions to aid their revision. True to the format of the examination, the MCQs are presented as six papers, with three each in basic sciences and clinical topics, and the short-answer questions come in groups of 20, so that the user may practice them against the clock. About a third of the MCQ answers receive annotation, and at least one reference source is always provided with the answers for both multiple-choice and short-answer questions. No comments are offered as to what level of scoring in these specimen papers might correlate with actual success in the examination.

The book should prove a useful aid as the questions appear to reflect accurately both the scope and level of papers that have been set. The referencing of question sources is particularly welcome, not only in helping the reader recognise how the ambiguity that inevitably attaches to some MCQs should be resolved in practice, but as a check against error. The references may even redeem the potentially cynical game of examination training if they prompt some reading beyond the sort in which information is valued according to how easily it can be boxed into question form. Several important papers are cited, and by choosing the Edinburgh and Oxford textbooks as the sources for the majority of their references, the authors seem to reflect common revision habits. (References are to the first edition of the Oxford book). Kaplan's textbook was repeatedly pillaged for material on dynamic psychiatry, although Rycroft's Penguin dictionary seems to have inspired question-setters in this area in the past. When the authors add their own comments these are typically helpful, with some bias towards clarification on neuroscientific points.

This is a well produced book that should prove useful to candidates for the Part II exam at all stages of revision. The authors' considerate notes lend it a distinct edge over several competitors.

C. J. MACE, *Senior Registrar in Psychiatry, St George's Hospital, London*

Schizophrenia: Positive and Negative Symptoms and Syndromes. Edited by N. C. ANDREASEN. Basel: Karger. 1990. 210 pp. £80.50.

The classification of schizophrenic symptoms into 'positive' and 'negative' has a history of only 10 years or so, which by the standard of most psychiatric concepts is very short indeed. In its brief life, however, it has managed to attract considerable attention from researchers.

This book summarises a decade's work on the topic. There are seven chapters: two by Andreasen on conceptual and methodological issues; one by Barnes & Liddle examining the validity of the classification; one by Marks & Luchins on brain imagery findings; one by Pickar *et al* on neurochemistry; one by Boyer *et al* on the efficacy of neuroleptics in patients with predominantly positive or negative symptoms; and one by McGlashan *et al* on the influence of psychosocial treatment on negative symptoms.

Altogether the book provides a comprehensive and well structured account of relevant research, and the contributions are well written. Thus, the reader should be able to come to their own conclusions about the value of the whole idea of positive and negative symptoms and syndromes. Most readers will probably conclude, like myself, that as with many psychiatric concepts it is good in parts.

On the negative side it can be criticised for not representing exactly what Hughlings Jackson meant. The attribution of 'positive' or 'negative' to some schizophrenic symptoms is at times questionable. The attempts to validate the system against radiological or biochemical criteria are rather unsatisfactory, and even the response of positive and negative symptoms to neuroleptics is not very predictable.

Despite all this, the system remains attractive. Why is this? I think that its appeal is most likely to be based on a fundamental principle of the mind's response to brain damage. This is that whenever one function or module (to use current jargon) is impaired, the overall effect is a composite of what the mind can no longer do *and* what the healthy modules do as a consequence. To take a neuropsychiatric example, someone who has sustained an acute cerebrovascular accident in the right frontoparietal region will no longer be able to move their left arm and leg (hemiplegia – a negative symptom) but at the same time may maintain that they *can* move these limbs (the delusion of anosognosia/denial – a positive symptom). The situation in schizophrenia is something akin to this, except that there are a host of things the mind can no longer do *and* a host of things that the mind starts doing as a consequence of each.

My main criticism of the positive–negative dichotomy is that it paints a false picture of how the so-called 'positive' symptoms originate. The mechanism proposed for a negative symptom – a failing or defunct module somewhere in the brain – is extremely plausible. However, I do not agree with the almost universally expressed opinion in this book about how delusions and hallucinations come about. Andreasen, for example, writes about positive symptoms: "We suspect that these patients may reflect an underlying structural abnormality in limbic regions which are abnormally "wired" to produce abnormal phenomena such as auditory hallucinations." In other words, positive symptoms develop out of an abnormal cerebral substrate to the same extent as negative symptoms do, and the abnormal substrate is *different* from that which causes negative symptoms. This is not generally true. Most instances of delusions and auditory hallucinations in schizophrenia are a *secondary* consequence on the part of a *normal* mind and brain to whatever causes the negative symptoms themselves. Only the negative symptoms are therefore intrinsically pathological.

This, of course, is a personal view, but the fact that I feel obliged to formulate it at least attests to the clarity of the arguments in this book.

JOHN CUTTING, *Consultant Psychiatrist, The Bethlem Royal Hospital, Beckenham*

Contemporary Approaches to Psychological Assessment. Edited by SCOTT WETZLER and MARTIN KATZ. New York: Brunner/Mazel. 1989. 348 pp. $40.00.

This book is one of a series of monographs published under the auspices of the Department of Psychiatry of the Albert Einstein College of Medicine. The aim of the series is to highlight developments in the field of psychiatry and especially to integrate clinical practice and research. The majority of contributors are members of this department, and the contents of this volume presumably reflect the diversity of interests and practices within it as well as the historical traditions of American psychiatry. The strong influences of psychoanalysis and projective tests are evident.

The book is divided into five sections: "Strategies of psychological assessment", which includes chapters on neuropsychological assessment and behavioural assessment of family functioning; "Specific methodologies", which consists of chapters covering structured interviews, self-report methods and projective tests; "Cognition and intellect", which covers intellectual assessments, neuropsychological tests and thought disorder; "Ego functioning"; and "Related issues", which includes chapters on cultural factors, physical illness and training. In all, there are 17 chapters; however, many of these are too short to give more than a superficial coverage of their subject area. This is a shame since some of the chapters are of great interest. Of particular note is the chapter by Blechman *et al* on the measurement of family interactions. Unfortunately, just as the reader becomes engrossed in this chapter it comes to an end.

The British reader, especially clinical psychologists, will find much of the material unlikely to reflect their interests or practice. The use of projective tests (especially the Rorshach), frequent reference to the Minnesota Multiphasic Personality Inventory (MMPI) and a rather stereotyped approach to the use of intellectual and neuropsychological tests could cast doubt on the use of the word 'contemporary' in the book's title. All in all, the brevity of many of the chapters, much of the subject matter and the price are unlikely to make this book attractive to many potential buyers.

NICHOLAS TARRIER, *Lecturer in Clinical Psychology, Department of Psychology, University of Sydney, Australia*

Conceptual Issues in Psychological Medicine. By MICHAEL SHEPHERD. London & New York: Tavistock/ Routledge. 1990. 335 pp. £40.00.

This is a collection of papers by one of the foremost intellects of modern psychiatry, published to mark the occasion of his retirement. It is the third in a series of collected papers and is something of a ragbag, although in with the rags are mixed some very elegant articles. Indeed, throughout the book the elegance of prose and breadth of thought is apparent in even the slightest of subjects, such as the review of Lieberman's *Biography of Otto Rank*.

The most entertaining chapter is the now well known Squibb lecture, "Sherlock Holmes and the case of Dr Freud". In this, Shepherd uses a plot similar to the '7% solution' to encase a detailed critique of the 'logic of psychoanalysis', likening it to the entirely spurious logic used by Holmes to solve his cases. Along the way we are treated to quotes from an extensive list ranging from Orson Welles to Peter Medawar, Morelli and Voltaire. Having heard the lecture and subsequently read the little pamphlet which was published, I could still not resist re-reading it.

The other major items include the classical work from 1961 on morbid jealousy which contains 80 case histories in illustration of the discussion. It was originally published in the *Journal of Mental Science*. How many journal editors would today accept such expansive justification for a clinical argument, however necessary? The Maudsley Lecture from 1987 "Changing disciplines in psychiatry" is another major work and "The legacies of Sir Aubrey Lewis" yet another. Here we begin to see one of the themes which connect the apparently disparate contributions together "Changing disiplines in psychiatry" is a title which is partly borrowed from the work of John Ryle, an early influence on Shepherd's career, whose biography forms one of the other chapters. The concern with wider social issues and with psychiatric epidemiology, including psychiatry in general practice, is apparent in several of the other chapters as would be expected. Shepherd's skill in combining these approaches with comments on classification of psychiatric illness and the use of psychotropic drugs is apparent in several of the other contributions.

The lighter contributions tend to be taken from book reviews and are for the most part brief appreciations of biographies of individuals such as Starobinski, Arise Evans and Karl Jaspers. The last chapter of all "What price psychotherapy?" is a good example of the uncompromisingly interrogative stance taken by Shepherd towards all claims to effectiveness by practitioners of various kinds of treatment which have not yet been justified. Although not contained in this volume, I was reminded of his attack on the early data for the efficacy of lithium. The point is not to take sides (for or against the treatment) but to indicate that there is room for reasonable doubt and to prompt greater clarity of thought and investigation. No doubt psychiatry will need other individuals to act as its super-ego now that Professor Shepherd has retired

CHRISTOPHER THOMPSON, *Professor of Psychiatry, University of Southampton, Southampton*

Advances in Psychology: 51. Progress in Reversal Theory. Edited by M. J. APTER, J. R. KERR and M P COWLES. Amsterdam: North-Holland. 1989. 394 pp $100.

Much of the book focuses on exploring the implications of Apter's reversal theory. Essentially, this proposes that our subjective experience is derived from two types of metamotivational states called telic (goal-orientated and future-directed) and paratelic (activity-focused). These are regarded as opposite and indeed the exploration of pairs of opposites is central to the theory. In the book it is noted how the operation of each of these two motivation states has advantages and disadvantages, how reversal (or switches) between these states occurs, how some personalities tend to be either telic (goal) or paratelic (activity) dominate, and how different behaviours and psychopathologies represent the operation of either telic or paratelic dominance. There is only one reference to Jung, although his concepts of personality types may have relevance.

The book contains six sections. The first offers four chapters on general themes. Apter himself gives a fascinating (if somewhat complex) account of emotion, making much of the distinction between somatic and transactional emotions and using the ideas of pairs of opposites in a way that will be familiar to affect theorists. The second section, "Stress and coping", contains seven chapters covering general issues of stress and type-A behaviour. Authors make the point that telic- and paratelic-dominate individuals experience and cope with stress in very different ways The third section contains six chapters on paradoxical behaviour (the persistence of behaviour despite strong evidence that it is damaging to self or others). Included here is a fascinating chapter by Braman on 'telic self-negativism' – the tendency to inhibit or act against one's own goals Section four has two chapters on states and reversals while the fifth section contains five chapters on dominance (not to be confused with the ethological concept) and its measurement. The final section, "Self and other", explores interpersonal themes and motives An appendix contains various self-report measures of the concepts discussed.

This book is a helpful overview of the new field of reversal theory. Not unusually, some chapters are better than others. In the preface the editors note that there has been concern with evidence for the theory and this book is partly to address this. I found the book stimulating and it certainly directed my thinking to an area of functioning that once stated seems in retrospect obvious Whether it will play a major role in how we conceptualise psychopathology only time will tell, but it would seem to hold promise. Various chapters explore stress and anxiety, delinquency, and to a lesser extent touch on depression. Whether it will advance our understanding of the biological basis of personality also remains an open question. In some of the studies comparison with neuroticism as a fear-based sensitivity would have been helpful; this may equally well have accounted for findings.

Reversal theory suffers the disadvantage of being a jargon-ridden theory, and at times it is difficult to remember what the different terms mean, although there is a helpful glossary Furthermore, for a book as expensive as this the production is appalling, with cheap word-processor typeset. While for some this will be a very helpful, new and fascinating approach, others may be less impressed.

PAUL GILBERT, *Top Grade Clinical Psychologist, Pastures Hospital, Derby*

Contents of
THE AMERICAN JOURNAL OF PSYCHIATRY

Volume 147, Number 9 September 1990

Presidential Papers: 1990

1113 Presidential address: defending humanistic values *Herbert Pardes*
1120 Response to the Presidential address: our children: our future. *Elissa P. Benedek*
1126 Herbert Pardes, MD, one hundred eighteenth President, 1989–1990 *H Keith H Brodie*

Special Article

1128 Family functioning and major depression· an overview *Gabor I. Keitner and Ivan W Miller*

Regular Articles

1138 Continuous versus targeted medication in schizophrenic outpatients· outcome results. *William T. Carpenter, Jr, Thomas E Hanlon, Douglas W Heinrichs, Ann T Summerfelt, Brian Kirkpatrick, Jerome Levine and Robert W. Buchanan*
1149 Neuroleptic malignant syndrome in 12 of 9792 Chinese inpatients exposed to neuroleptics. a prospective study. *Ming Zhen Deng, Guo Qiang Chen and Michael R Phillips*
1156 Factors in the development of severe forms of tardive dyskinesia. *Ramzy Yassa, Neelakanta P V Nair, Hani Iskandar and George Schwartz*
1164 Cocaine abuse among schizophrenic patients *Kathleen Brady, Raymond Anton, James C Ballenger, R. Bruce Lydiard, Bryon Adinoff and Jean Selander*
1168 Maladaptive denial of physical illness. a proposal for *DSM–IV David H Strauss, Robert L. Spitzer and Philip R. Muskin*
1173 Variability in the application of contemporary diagnostic criteria: endogenous depression as an example *Mark Zimmerman, William H Coryell and Donald W. Black*
1180 Regional variation in patterns of inpatient psychiatric care *Robert Rosenheck and Boris Astrachan*
1184 Relation of clinical variables to dissociative phenomena in eating disorders. *Mark A. Demitrack, Frank W Putnam, Timothy D Brewerton, Harry A. Brandt and Philip W Gold*
1189 Time-related predictors of suicide in major affective disorder. *Jan Fawcett, William A. Scheftner, Louis Fogg, David C Clark, Michael A. Young, Don Hedeker and Robert Gibbons*
1194 Life events and the course of bipolar disorder *Aimee Ellicott, Constance Hammen, Michael Gitlin, Gary Brown and Kay Jamison*
1199 Comorbidity of major depression and anxiety disorders in twin pairs. *Svenn Torgersen*
1203 Amoxapine versus amitriptyline combined with perphenazine in the treatment of psychotic depression. *Raymond F Anton, Jr and Earl A Birch, Jr*
1209 A controlled trial of fluvoxamine in obsessive-compulsive disorder: implications for a serotonergic theory. *Michael A. Jenike, Steven Hyman, Lee Baer, Amy Holland, William E. Minichiello, Lynn Buttolph, Paul Summergrad, Ralph Seymour and Joseph Ricciardi*
1216 Alexithymic features in relation to the dexamethasone suppression test in a Finnish population sample. *Tomi Lindholm, Ville Lehtinen, Markku T. Hyyppa and Pauli Puukka*

Commentary

1220 The coming crisis in funding child psychiatry training. *Robert S. McKelvey*

Clinical and research reports

1225 CSF 5–HIAA and HVA concentrations in elderly depressed patients who attempted suicide *J Sidney Jones, Barbara Stanley, J. John Mann, Allen J. Frances, Jeannine R. Guido, Lil Traskman-Bendz, Ronald Winchel, Richard P Brown and Michael Stanley*
1228 A preliminary investigation of alexithymia in men with psychoactive substance dependence. *Graeme J. Taylor, James D. A. Parker and R. Michael Bagby*
1231 Prevalence of neuroleptic-induced dystonia in mania and schizophrenia. *Gary J Remington, George Voineskos, Bruce Pollock, Ken Reed and Kathryn Coulter*
1234 Positive and negative symptoms and social competence in adolescents at risk for schizophrenia and affective disorder. *Robert H. Dworkin, Susan R. Green, Nancy E. Small, Matthew L Warner, Barbara A. Cornblatt and L. Erlenmeyer-Kimling*

Contents of The Journal of Nervous and Mental Disease

September 1990 **Volume 178, Number 9**

The neural basis of the deficit syndrome of schizophrenia. *Brian Kirkpatrick and Robert W. Buchanan* 545

Some biological and behavioural features associated with clinical personality types. *Ronald J. Gurrera* 556

Treatment of panic and agoraphobia. An integrative review. *Richard P. Mattick, Gavin Andrews, Dusan Hadzi-Pavlovic and Helen Christensen* 567

Comparison of early and late onset dysthymia. *James P. McCullough, James A. Braith, Rees C. Chapman, Matthew D. Kasnetz, Karen F. Carr, James H. Cones, Jeffrey Fielo, Owen S. Shoemaker and William C. Roberts* 577

Duration and intensity of combat exposure and posttraumatic stress disorder in Vietnam veterans. *L. Buydens-Branchey, D. Noumair and M. Branchey* 582

A description of patients diagnosed with DSM-III-R generalized anxiety disorder. *William C. Sanderson and David H. Barlow* 588

An examination of defense style in parents who abuse children. *John Brennan, Gavin Andrews, Allen Morris-Yates and Christopher Pollock* 592

Brief Reports

Comparison of dysthymic major and nonmajor depressives 596

Familiality of DSM-III self-defeating personality disorder 597

Triiodothyronine augmentation of the antidepressant effect of the nontricyclic antidepressant trazodone 598

Book Reviews. Beisser, A. *Flying Without Wings;* Sichrovsky, P. *Born Guilty: Children of Nazi Families;* Lewes, K. *The Psychoanalytic Theory of Male Homosexuality;* Miller, T. W. (ed.) *Stressful Life Events;* McKinney, W. T. (ed.) *Models of Mental Disorders: A New Comparative Psychiatry;* Stotland, N. L. *Social Change and Women's Reproductive Health Care: A Guide for Physicians and Their Patients;* Brumberg, J. J. *Fasting Girls: The Emergence of Anorexia Nervosa as a Modern Disease;* Goodwin, D. W. *Alcohol and the Writer;* Herschman, D. J. and Lieb, J. *The Key to Genius: Manic Depression and the Creative Life;* Levin, H. S., Eisenberg, H. M. and Benton, A. L. *Mild Head Injury* 600

Index to Advertisers

Pharmaceuticals

The Boots Company plc .... *facing* p 480
Duphar Laboratories .... *facing* p 481
Janssen Pharmaceuticals Ltd. .... p vi
Janssen Pharmaceuticals Ltd. .... *facing* p 529
Janssen Pharmaceuticals Ltd. .... *facing* p 532
Lundbeck Limited .... inside front cover
Lundbeck Limited .... outside back cover
E. Merck Ltd. .... *facing* p 528
Eli Lilly .... *facing* p 522
Eli Lilly .... *facing* p 523

Appointments

Dr. Michael Craft .... *facing* p 523
Langley Memorial Hospital .... p iv
Yarmouth Regional Hospital .... p iv

Booksellers & Publishers

Oxford University Press .... p v

Clinics, Hospitals & Nursing Homes

St. Andrew's Hospital .... p ii

Printed in Great Britain by Henry Ling Ltd., at the Dorset Press, Dorchester, Dorset

STORMS BLOW
FIND THE EYE
WINDS CHANGE
... REFLECT (Anon)

CLOPIXOL®
NEW ACUPHASE
zuclopenthixol acetate ▼

A harmonious new way to treat acute psychosis

Lundbeck

FACING THE FUTURE TOGETHER

Further information is available on request from:

20 DEC 19